Обзор / Review

Allergic rhinitis in children with obesity: a modern view of the problem

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Abstract

Relevance. In recent decades, there has been an increase in a number of non-communicable chronic diseases and treated as a global health priority. There is an increase in the prevalence of allergic diseases, including allergic rhinitis (AR), and obesity in the pediatric population. In this regard, the study of AR in children with comorbid obesity is of particular interest.

The aim of the review is to summarize current data on the immunological and clinical-epidemiological features of AR in children with comorbid obesity.

Content. The review presents current information on the role of individual cytokines and adipokines in the development of chronic systemic inflammation in children with AR and obesity. An analysis of literature data on the significance of obesity as a possible risk factor for the development of AR in childhood is conducted. Clinical and epidemiological features are discussed, and individual studies are presented on some aspects of AR therapy in obese patients.

Conclusions. The analysis showed that the currently available data on the relationship between AR and overweight/obesity in children are contradictory and require further research.

Keywords: allergic rhinitis, obesity, cytokines, interleukin-33, interleukin-1β, leptin, adiponectin

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Аллергический ринит у детей с ожирением: современный взгляд на проблему

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Резюме

Актуальность. В последние десятилетия отмечается эпидемический рост ряда неинфекционных хронических заболеваний, являющихся глобальной проблемой здравоохранения. В детской популяции наблюдается увеличение распространенности как аллергического ринита (АР), так и ожирения. В связи с этим представляет особый интерес изучение АР у детей с коморбидным ожирением.

Целью обзора является обобщение современных данных об иммунологических и клинико-эпидемиологических особенностях АР у детей с коморбидным ожирением.

Содержание. В обзоре представлены актуальные сведения о роли отдельных цитокинов и адипокинов в формировании хронического системного воспаления у детей с АР на фоне избытка жировой массы тела. Проведен анализ литературных данных о значении ожирения как возможного фактора риска развития АР в детском возрасте. Обсуждены клинико-эпидемиологические особенности, а также приведены единичные исследования, касающиеся некоторых аспектов терапии АР у пациентов с ожирением.

Заключение. Проведенный анализ показал, что имеющиеся на сегодняшний день данные о наличии связи АР и избытка массы тела / ожирения у детей носят противоречивый характер и требуют проведения дальнейших исследований.

Ключевые слова: аллергический ринит, ожирение, цитокины, интерлейкин-33, интерлейкин-1β, лептин, адипонектин

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INTRODUCTION

In recent decades, the epidemic growth of allergic diseases has become a serious problem for global health [1]. AR is one of the most common chronic diseases in the world and is associated with a significant deterioration in the quality of life of patients [2]. Risk factors for AR include genetic predisposition, high socioeconomic status, maternal smoking during the first year of a child's life, and a history of cesarean section [2]. At the same time, recent studies show that excess body fat in children can be considered one of the predictors of AR development [3]. It is assumed that the link between excess body weight/obesity and AR is due to common pathophysiological mechanisms [3, 4]. Obesity is characterized by hypertrophy of white adipose tissue and impaired metabolic activity of adipocytes, leading to chronic systemic inflammation [4, 5]. In turn, changes in the levels of adipokines secreted by adipose tissue can cause a shift in the immune response toward the Th2 type, which increases the risk of allergic diseases [4, 6, 7].

It should be noted that modern literature provides contradictory data regarding the link between obesity and AR [3, 8]. Thus, it is important to summarize current data on the possible effect of obesity on the development of AR and the characteristics of the disease, as well as on the mechanisms underlying these processes.

PATHOGENETIC FEATURES OF ALLERGIC RHINITIS IN CHILDREN WITH OBESITY

It is assumed that there is some commonality between obesity and AR in terms of pathophysiological mechanisms, in particular, changes in the production of pro- and anti-inflammatory cytokines and adipokines [9, 10]. However, the exact mechanism explaining the possible link between obesity and AR remains unclear [7,10].

Leptin is one of the hormones produced by adipose tissue that regulates energy metabolism. Leptin promotes the production of cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), interleukin-12 (IL-12), and stimulates the proliferation of Th1 cells [10]. Recent data indicate that patients with RA and comorbid obesity have elevated serum leptin levels, which correlate with the severity of RA [11–15, 23].

According to Wang X. et al., children with AR and obesity had a significant increase in leptin and ILC2 (type 2 innate lymphoid cells) levels in the blood compared to children with AR and normal body weight, as well as the control group (healthy children). It has been shown that the administration of recombinant leptin to children with AR and obesity led to an increase in ILC2 levels in the blood [11]. It is worth noting that similar results were obtained in another study in adult patients with AR who were

obese [12]. According to the authors, the leptin/ILC2 axis in obese patients may exacerbate chronic systemic inflammation and contribute to the development of severe AR [11, 12]. It is assumed that leptin enhances the activation of inflammatory cells in AR against the background of obesity. In a study by Liu W. et al., elevated serum leptin levels in children with AR were positively correlated with levels of eosinophils, eosinophil cationic protein (ECP), C-reactive protein (CRP), interleukin-5 (IL-5), and interleukin-17 (IL-17) [13].

The possible role of osteopontin protein in allergy and obesity is discussed. Osteopontin is a pro-inflammatory cytokine secreted by osteoblasts, fibroblasts, epithelial cells, activated macrophages and T-lymphocytes [16]. A number of studies have shown that in children with AR and obesity there is a significant increase in the level of leptin and osteopontin in the serum of rabbits compared to the control group, as well as a positive correlation with the severity of AR. The level of osteopontin was found to correlate with the level of eosinophils and ECB in serum. Thus, increased expression of leptin and osteopontin may play an important role in the pathogenesis of chronic inflammation in AR and morbid obesity, contributing to the enhancement of Th2 response, as well as regulating apoptosis, adhesion, migration and activation of eosinophils [16, 17].

It should be noted that there are studies in the literature that did not reveal statistically significant differences between leptin levels and the severity/character of AR course in obese patients [18, 19]. Thus, a cross-sectional study showed that in 7-10 years old overweight/obese children (according to bioimpedance-sometry data) no significant differences in serum leptin levels were found depending on the nature of AR course (persistent/intermittent) [18]. In a study by Kalpa- klioglu A.F. et al. there was no association between serum leptin levels and the severity of AR, as well as sensitization to allergens in adult patients [19].

Altered adiponectin secretion possibly contributes to the development of chronic systemic inflammation in patients with AR and comorbid obesity. However, there are very few studies devoted to this problem [4, 7]. It is known that in obesity there is a decrease in the level of adiponectin, an adipose tissue hormone with an anti-inflammatory effect [5, 7]. n an experimental study on a mouse model, it was shown that exogenous administration of adiponectin attenuated the development of ovalbumin-induced airway hyperreactivity by reducing the number of eosinophils and decreasing the level of Th2-type cytokines [22]. In a cross-sectional study, it was found that children 4-10 years old with first-diagnosed AR had lower serum adiponectin levels compared to healthy controls and positively correlated with the severity of AR. Adiponectin levels were negatively correlated with serum ECB levels, while there was no correlation with interleukin-1β (IL-1β), IL-6, TNF-α, IL-10 and total IgE levels [14]. In another study, serum adiponectin levels in children 7-10 years old with AR and obesity had no significant differences compared to the control group. Comparative analysis of adiponectin levels in children with AR and obesity did not reveal statistically significant differences depending on the nature of AR course [18].

Interleukin-1β (IL-1β) is considered as another mediator of allergic inflammation. In AR, IL-1β levels may be associated with inflammation caused by exposure to allergens, as well as with the main clinical symptoms of the disease - nasal itching and rhinorrhea [21]. It should be noted that there are single clinical studies in the literature devoted to the study of the role of IL-1β in AR on the background of excess adipose tissue. According to a number of authors, IL-1β can be a potential biomarker and predictor of severe persistent AR [22, 23, 24]. According to Han M.W. et al. data, IL-1β level positively correlates with AR severity and is also a significant risk factor for the development of moderate and severe persistent AR (4.7-fold increase in risk) [22]. According

to another study, children with AR and obesity have higher IL-1β levels compared to children with AR and normal body weight. It is shown that elevated IL- 1β level (5.8-fold increase in risk) and elevated leptin level (11.3-fold increase in risk) are significant risk factors for moderate to severe persistent AR. The authors found that the majority of children with AR have increased serum IL-1β and leptin levels during weight gain. In turn, in some children with obesity and AR, a decrease in blood levels of IL-1β and leptin was observed during weight correction [23]. In another study, the authors showed that in the group of obese children IL-1β level depends on the nature of AR course, namely IL-1β level in children with persistent AR was significantly higher compared to children with intermittent AR course [24].

Interleukin-33 (IL-33) is a cytokine "alarmin" (alarm signal) that can accumulate and be rapidly released into the extracellular space during cell/tissue damage, affecting immune cells expressing ST2 receptor on their surface [25, 26]. IL- 33 is synthesized mainly by epithelial cells, fibroblasts, endothelial cells, and adipose tissue cells - adipocytes. It is known that IL-33 is produced by the cells of the first line of defense, in particular epithelial cells, when they are exposed to allergens [27, 28]. In vitro studies have shown that IL-33/ ST2 causes activation of ILC2, which induce Th2 response and tissue remodeling by producing Th2-type cytokines [29, 30]. In adipose tissue, IL-33 is involved in the interaction between adipocytes and immune cells [31]. Exposure of IL-33 to ILC2 cells leads to their activation and secretion of IL-5, IL-13. In turn, IL-13 directly affects pre-adipocytes and promotes their maturation [32]. IL-33 interacts with Treg having ST2-receptor on its surface, which causes synthesis of IL-10 by these cells, which causes alternative activation of macrophages and their synthesis of catecholamines aimed at maturation of pre-adipocytes into adipocytes [32, 33]. According to Glück J. et al., adult patients with intermittent seasonal AR have elevated serum IL-33 levels that correlate with the severity of AR [32]. In a cross-sectional study, it was shown for the first time that children 7-10 years old with overweight/obesity and AR showed increased serum IL-33 levels compared to the control group. In children with excess body fat mass the IL-33 level depended on the nature of AR course, in particular, in children with persistent AR the IL-33 content was significantly lower compared to children with intermittent AR. An inverse correlation between IL-33 level and %FMT was found to be lower in intermittent AR compared to children with intermittent AR [24].

Thus, few data from the literature suggest a possible role of cytokines IL1β, IL-33, osteopontin and adipokines (leptin, adiponectin) in AR and comorbid obesity in children. However, further prospective studies are needed to clarify the potential mechanisms of the association between AR and pediatric obesity.

IS OBESITY A RISK FACTOR FOR ALLERGIC RHINITIS: WHAT IS KNOWN?

Currently, there is no consensus on the role of overweight/obesity as a risk factor for the development of AR (Table 1). A large number of studies indicate an association between high BMI and the risk of AR development in children [34-37]. It is important to note that the cross-sectional and retrospective design of these studies does not allow assessing whether increased BMI preceded the development of AR in children.

There are few examples of prospective studies on this topic in the current literature. For example, Vehapoglu A. et al. found that prepubertal obese children have a higher risk of developing AR and BA compared to children with normal body weight [38]. In another study, an increase in BMI in children aged 16-18 years was associated with an increased risk of developing AR, but not BA and AD [39].

Few studies are available to assess the dynamics of body weight after birth and its possible impact on the future development of AR in children. In a retrospective study, excessive weight gain after birth was found to be associated with the risk of developing AR and atopic dermatitis (AD) in adolescence, especially in children born with low birth weight [40].

However, Mai X.M. et al. reported that no association between large birth weight (⩾90th percentile) and the presence of AR and AD symptoms was found in 4-year-old children [41]. Chang C.L. et al. studied the association between BMI after birth and the risk of allergic diseases (AR, food allergy) in children aged 6, 12, and 18 years.The risk factors for AR in children were found to be delayed fetal intrauterine development, insufficient weight gain, maternal heredity for allergy, but not excessive weight gain during the first two years of life [42].

In a meta-analysis of 30 studies, high BMI was associated with an increased risk of AR in children but not in adults [3]. In another systematic review and metaanalysis of 32 studies, high or low BMI was not associated with the risk of developing AR in both children and adults [8]. However, in this paper [8], unlike the previous meta-analysis [3], data in children were evaluated in age-dependent subgroups: under 12 years of age and between 13-18 years of age. It is worth noting that both meta-analyses included studies based on self-reports of AR symptoms and self-reported anthropometric measurements, which could influence the authors' conclusions in a different way [3, 8].

There are also data in the literature in which excess body fat mass in children was not a risk factor for AR [43-47]. According to the results of phase II studies, ISAAC ("International study of asthma and allergies in childhood") involving 10,652 children 8-12 years of age found no association between high BMI and AR [43]. In several cross-sectional studies in children, high BMI had a negative association with AR [44, 45]. A study by Scaaby T. et al. using Mendelian randomization found an association between overweight/obesity and higher prevalence of AD, decreased lung function in patients older than 16 years, but not with AR [46]. According to the data presented by Han Y.Y. et al. in children, central obesity is associated with a reduced risk of AR [47].

The ambiguity of the study results is probably related to the design features, as well as to the different methods of AR verification and overweight/obesity in children. It is worth noting that the literature on this problem is dominated by studies with a cross-sectional or retrospective design, which do not allow to determine the presence of causality [33-37, 40, 43, 44, 46, 47]. In some studies, the criterion for children inclusion was the presence of a physician-verified diagnosis of AR [33, 37, 38, 41, 42], whereas in other studies the assessment of AR symptoms was based only on parent/child questionnaire data [36, 39, 40, 44, 47]. A number of studies analyzed self-reports of patients and/or parents of children, which served to further identify groups of children for additional allergologic examination and confirmation of AR diagnosis [34, 35, 43, 46]. The children's anthropometric indicators at the time of their inclusion in the study were assessed both by medical personnel [33- 35, 37, 38, 41, 43, 44, 47] and parents [39, 36, 40, 42, 46]. All this causes certain difficulties when analyzing scientific works on this topic.

Thus, there are few and rather contradictory studies investigating the possible role of overweight/obesity as a risk factor for AR in children. It is still unclear which values of increased BMI are associated with the development of AR, since both overweight and obese children were included in the studies. It should be noted that in most studies the presence of excess body fat mass in children was diagnosed using the BMI criterion [33-46], which does not allow determining the actual distribution of body fat [48, 49]. According to a meta-analysis, BMI is characterized by low sensitivity and does not identify more than a quarter of overweight/obese children [49]. This raises the question of whether further studies investigating AR in children with excess body fat mass should use the generally accepted BMI or use other more accurate methods of assessing body composition. In addition, the propensity of obese children to develop AR is likely to be influenced by interactions with other factors such as sex, race, aggravated heredity and the presence of comorbid allergic diseases, which should be considered in future studies.

PECULIARITIES OF CLINICAL SYMPTOMATOLOGY AND PREVALENCE OF ALLERGIC RHINITIS IN CHILDREN WITH OBESITY

It is of interest to study the clinical features of AR in children with excess body fat mass. Thus, in

Table 1. **Results of clinical studies in children with allergic rhinitis and obesity (authors' table)**
Таблица 1. **Результаты клинических исследований у детей с аллергическим ринитом и коморбидным ожирением (таблица автора)**

Author, year	Country	Type of study	Sample size	Age of children	Main outcome
Lei Y., et al., 2016 [33]	China	Cross-sectional study	3327 children	3 age groups: 2–6, 7–12, 13–14	Obesity in children is a risk factor of AR and AD, but not BA, FA and DA
Saadeh D. et al., 2014 [34]	France	Cross-sectional study	6733 children	9–11	High BMI is a risk factor for AR and BA
Baumann L.M. et al., 2015 [35]	Pery	Cross-sectional study	1441 children	13–15	High BMI is a risk factor for AR
Lim M.S. et al., 2017 [36]	South Korea	Cross-sectional study	53 769 children	12–18	High BMI is associated with AR and AD
Vehapoglu A., et al., 2021 [38]	Turkey	Prospective study	707 children	3–10	Overweight/obesity is associated with a high risk of AR and BA
Kreißl S. et al., 2014 [39]	Germany	Prospective study	3000 children	Evaluation at the age of 9–11 and 16–18	Increase in BMI is associated with a high risk of AR, but not BA and AD
Lin M.H. et al., 2015 [40]	Taiwan	Retrospective study	74 688 children	13–15	Excessive body weight gain. after birth may be a risk factor for AR and AD development
Mai X.M., et al., 2007 [41]	Sweden	Prospective study	4089 children	Evaluation at the age of 1, 2 and 4	Birth weight ⩾ 90th percentile and high BMI in early childhood are not a risk for AR at the age of 4
Chang C.L., et al., 2022 [42]	Australia	Prospective study	620 children	Evaluative at the age of 6, 12 and 18	Excess body weight is not associated with a risk of AR at the age of 18
T. Kusunoki et al., 2007 [44]	Japan	Cross-sectional study	50 086 children	7–15	The inverse association is between childhood obesity and prevalence of AR and AC. Obesity in children is associated with prevalence of BA and severity of AD
Leung T.F., et al., 2009 [45]	China	Cross-sectional study	486 children	14–16	Obesity is not associated with AR and BA
Y.Y. Han et al., 2007 [47]	USA	Cross-sectional study	2358 children	6–17	Central obesity is associated with a decreased risk of AR, particularly among boys

Note: FA — food allergy, DA — drug allergy, AC — allergic conjunctivitis.

some studies, the authors demonstrated the presence of an association between obesity and the severity of AR [13, 18, 24, 39]. According to a prospective study of 1794 children observed from 9-11 years to 16-18 years, an increase in BMI was a risk factor for persistent AR [50]. In the work of Han M.W. it was shown that in some children with mild AR and normal body weight on the background of weight gain there was observed aggravation of nasal symptoms and development of severe AR [23]. At the same time, in the work of Kusunoki T. et al. no association was found between obesity and severity of AR in children. In this paper, the presence of AR was assessed by parental questionnaires and patients with other allergic diseases (AD, AD, AU) were included in the study [44].

It should be noted that in the vast majority of studies, the authors used BMI to assess obesity, which does not distinguish between fat and muscle mass in the body, nor does it take into account the distribution of fat deposits [49, 50]. In a few studies, the presence of obesity was assessed by bioimpedanceometry [13, 18]. In particular, by the % body fat

mass (%BFM), which reflects the presence and degree of fat deposition in the body [51, 52]. Thus, Liu W. et al. found that in 3126 children aged 7-12 years, obesity (according to %BFM, waist circumference, and BMI) was a risk for severe year-round AR [13]. According to a cross-sectional study, 7-10 years old overweight/obese children (according to %BFM) had an earlier manifestation of AR (up to 3 years of age) with nasal pruritus predominating as the main manifestation of rhinitis. Excess body fat mass was a predictor of persistent AR course in primary school children [18].

Children with AR and comorbid obesity may be more susceptible to air pollution. The results of Li R. L. et al. indicate that carbon monoxide (CO) and ambient particles less than 10 μm in diameter (PM10) and PM2.5 lead to worsening nasal symptoms of AR in obese children compared to the ones of control group [53].

It is of interest to study the possible role of excess body fat mass in the formation of a certain type of sensitization. According to a study, children 4-8 years old with AR and increased BMI were 2.64 times more likely to be monosensitized to aeroallergens of house dust mites compared to children with AR and normal BMI [54]. It has also been shown that skin reactivity to histamine in adult patients with AR may depend on body fat mass. According to a study by Park D. Y. et al. observed a positive correlation between high BMI and skin reactivity to histamine in patients with AR [55]. In experimental studies it was shown that histamine level can be increased as a result of the release of substance P, which leads to neurogenic inflammation [56, 57]. Thus, according to the authors, the increased content of substance P may be a mediator of allergy, providing higher skin reactivity to histamine in obesity [55, 56, 57].

A number of epidemiologic studies have established higher prevalence rates of AR among overweight/obese children compared to children with normal body weight [33, 35, 37]. At the same time, Liu W. et al. found that the prevalence of obesity in the group of children with AR was comparable to children in the control group [13].

In addition, children with AR have a pronounced decrease in quality of life parameters related to physical activity. According to Park J. H. et al., children with AR symptoms (rhinorrhea, nasal congestion, sneezing, nasal itching, sleep disturbance) are more prone to sedentary lifestyle than AD patients and control group children. At the same time, higher BMI values were observed in the group of children with AR at the time of the study compared to children in the control group. According to the authors, children with AR have a higher probability of obesity. It should be noted that in the study, the level of physical activity was assessed based on children's self-reported physical activity rather than objective measurements (e.g., accelerometer), which may have led to underestimation or overestimation of the relationship between the level of physical activity, BMI and AR [58].

Given that most studies have a cross-sectional design, it remains unclear whether excess body fat mass/obesity in children precedes the development of the identified clinical features of AR. On the other hand, it is possible that patients with certain features of AR are more likely to gain weight. To answer these questions, further prospective studies using strict diagnostic criteria for AR and the use of methods to assess the presence and degree of body fat deposition are needed. In addition, epidemiologic studies are needed to assess the prevalence of AR in children of different ages with comorbid obesity.

TREATMENT OF PATIENTS WITH ALLERGIC RHINITIS AND OBESITY

Despite the fact that the principles of therapy have not been developed for individuals with AR and obesity, single studies have demonstrated var-

ious aspects of treatment in these patients [23, 59]. According to the data of Han M.W. et al., improvement of nasal symptoms from severe to mild AR was observed in a part of children on the background of BMI reduction [23]. At present, it is difficult to say whether body fat mass affects the response of patients with AR to treatment with intranasal glucocorticosteroids (INGCS). In a recent prospective study of the efficacy of INGCS in adult patients with AR and elevated BMI, it was shown that symptom improvement was observed after a 30-day course of treatment in both obese and normal weight patient groups. AR symptoms were assessed using the Visual analogue scale (VAS), SNOT- 22 (Sino-Nasal Outcome Test-22) and NOSE-5 (Nasal obstruction symptom evaluation-5) questionnaires, as well as an increase in peak nasal inspiratory flow (PNIF). At the same time, the authors showed that patients with AR and obesity had a statistically significant decrease in IL-10 levels in the nasal mucosa after therapy compared to the group with AR and normal body weight. Thus, according to the authors' opinion, individuals with AR and obesity have a weaker anti-inflammatory response to INGCS therapy [59].

Thus, no studies on the efficacy of INGCS in children with AR and obesity were found in the literature available to us. This leads to another unanswered question: whether weight loss in obese children can improve the results of AR treatment with the use of INGCS. In general, there is insufficient work on the role of weight loss as an additional factor in influencing symptoms and efficacy of AR treatment in children. Further study of the impact of obesity on the treatment of children with AR, as well as weight correction is important from the point of view of personalized tactics of managing such patients.

CONCLUSION

Obesity is characterized by chronic sluggish inflammation, which can influence the immune system and possibly contribute to the pathogenesis of AR. To date, there is no consensus on which of the potential mechanisms mediates the association between AR and obesity. Summarizing these studies, we can conclude that patients with AR and excess body fat mass have an imbalance of IL-33, IL-1β, osteopontin, and a number of adipokines (leptin, adiponectin). Given the limited data, it is currently difficult to say which of these are most clinically important.

At present, the question of whether overweight/obesity is a risk factor for AR in children remains incompletely resolved. There is evidence that obesity may affect the severity of AR. At the same time, there are a number of studies in the literature that have not found an association between these diseases.

In general, studies on the causal relationship between obesity and AR are few and contradictory. In our opinion, it is of particular interest to continue studying the role of cytokines and adipokines in AR on the background of excess body fat mass. In addition, it remains unclear whether excess body fat mass/obesity in children precedes the development of the identified clinical features of AR, or whether patients with certain clinical features are more likely to gain weight. Future work should include studies related to weight management to assess the clinical and pathophysiologic significance of the putative mechanisms underlying the association between AR and excess body fat mass. It is of interest to study the impact of obesity on the therapy of children with AR, as well as to evaluate whether weight management can improve the response to INGCS treatment in children with AR. All this calls for further in-depth study of AR in overweight/obese children.

REFERENCES

1. Passali D., Cingi C., Staffa P. et al. The International Study of the Allergic Rhinitis Survey: outcomes from 4 geographical regions. Asia Pacific Allergy. 2018; 8 (1): 1–15. https://doi.org/10.5415/apallergy.2018.8.e7.
2. Bousquet J., Anto J.M., Bachert C. et al. Allergic rhinitis. Nature reviews. Disease primers. 2020; 6 (1): 95. https://doi.org/10.1038/s41572-020-00227-0.
3. Zhou J., Luo F., Han Y. et al. Obesity/overweight and risk of allergic rhinitis: A meta-analysis of observational studies. Allergy: European Journal of Allergy and Clinical Immunology. 2020; 75 (5): 1272–1275. https://doi.org/10.1111/all.14143.
4. Kelishadi R., Roufarshbaf M., Soheili S. et al. Association of Childhood Obesity and the Immune System: A Systematic Review of Reviews. Childhood Obesity. 2017; 13 (4): 332–346. https://doi.org/10.1089/chi.2016.0176.
5. Migacheva N.B., Skvortsova O.V., Kaganova T.I., Ginzburg A.S. Paradoxes of the immune response in obesity. Effective Pharmacotherapy. 2023; 19 (28): 30–36. (In Russ.)
6. Recinella L., Orlando G., Ferrante C. et al. Adipokines: New Potential Therapeutic Target for Obesity and Metabolic, Rheumatic and Cardiovascular Diseases. Frontiers in physiology. 2020; 11: 1–32. https://doi.org/10.3389/fphys.2020.578966.

7. Umano G.R., Pistone C., Tondina E. et al. Pediatric Obesity and the Immune System. Frontiers in Pediatrics. 2019; 7: 1–9. https://doi.org/10.3389/fped.2019.00487.
8. Yeo B.S.Y., Guan E.J., Ng K. et al. Association of Abnormal Body Weight and Allergic Rhinitis-A Systematic Review and Meta-Analysis. Clinical and Experimental Allergy. 2025; 55 (2): 142–165. https://doi.org/10.1111/cea.14604.
9. Tajima H., Pawankar R. Obesity and adiposity indicators in asthma and allergic rhinitis in children. Current opinion in allergy and clinical immunology. 2019; 19 (1): 7–11. https://doi.org/10.1097/ACI.0000000000000504.
10. Stefani C., Pecoraro L., Flodmark C.E. et al. Allergic Diseases and Childhood Obesity: A Detrimental Link? Biomedicines. 2023; 11: 2061. https://doi.org/10.3390/biomedicines11072061.
11. Wang X., Shen Y., Ke X. et al. Role of leptin/ILC2 axis in allergic rhinitis in obese children. International journal of pediatric otorhinolaryngology. 2022; 157. https://doi.org/10.1016/j.ijporl.2022.111127.
12. Wang X., Hu G., Shen Y. et al. Leptin mediated activation of group 2 innate lymphocytes aggravates the pathogenesis of allergic rhinitis in obese adults. 2022; 38 (12): 1118–1124.
13. Liu W., Zeng Q., Zhou L. et al. Association of leptin with disease severity and inflammation indicators in Chinese obese children with allergic rhinitis. Pediatric allergy and immunology. 2018; 29 (2): 186–193. https://doi.org/10.1111/pai.12856.
14. Hsueh K.C., Lin Y.J., Lin H.C. et al. Serum leptin and adiponectin levels correlate with severity of allergic rhinitis. Pediatric allergy and immunology: official publication of the European Society of Pediatric Allergy and Immunology. 2010; 21 (1): 155–159. https://doi.org/10.1111/j.1399-3038.2009.00878.x.
15. Unal M., Eskandari G., Muşlu N. et al. Serum leptin levels in patients with allergic rhinitis. Otolaryngology-head and neck surgery: official journal of American Academy of Otolaryngology-Head and Neck Surgery. 2006; 134 (2): 331–333. https://doi.org/10.1016/j.otohns.2005.11.021.
16. Zeng Q., Luo X., Han M. et al. Leptin/Osteopontin Axis Regulated Type 2T Helper Cell Response in Allergic Rhinitis with Obesity. Ebiomedicine. 2018; 32: 43–49. https://doi.org/10.1016/j.ebiom.2018.05.037.
17. Liu W., Zeng Q., Chen Y. et al. Role of leptin/osteopontin axis in the function of eosinophils in allergic rhinitis with obesity. Mediators of Inflammation. 2018: 1–10. https://doi.org/10.1155/2018/9138904.
18. Koroleva A.E., Bekezin V.V., Meshkova R.Ya., Demina E.G., Tsvetnaya I.N. Phenotypic markers of allergic rhinitis in children with overweight and obesity. Russian Bulletin of Perinatology and Pediatrics. 2023; 68 (4): 351. (In Russ.)
19. Kalpaklıoglu A.F., Baccioglu A., Yalim S.A. Does serum leptin levels differ between patients with rhinitis of allergic versus nonallergic etiology? The clinical respiratory journal. 2021; 15 (12): 1352–1358. https://doi.org/10.1111/crj.13440.
20. Shore S.A., Terry R.D., Flynt L. et al. Adiponectin attenuates allergen-induced airway inflammation and hyperresponsiveness in mice. The Journal of allergy and clinical immunology. 2006; 118 (2): 389–395. https://doi.org/10.1016/j.jaci.2006.04.021.
21. Wang H.R., Wei S.Z., Song X.Y. et al. IL-1β and Allergy: Focusing on Its Role in Allergic Rhinitis. Mediators of Inflammation. 2023: 1265449. https://doi.org/10.1155/2023/1265449.
22. 22. Han M.W., Kim S.H., Oh I. et al. Serum IL-1β can be a biomarker in children with severe persistent allergic rhinitis. Allergy Asthma Clin Immunol. 2019; 18 (15): 58. https://doi.org/10.1186/s13223-019-0368-8.
23. Han M.W., Kim S.H., Oh I. et al. Obesity Can Contribute to Severe Persistent Allergic Rhinitis in Children through Leptin and Interleukin-1β. International Archives of Allergy and Immunology. 2021; 182 (6): 546–555. https://doi.org/10.1159/000512920.
24. Koroleva A.E., Bekezin V.V., Sergeeva I.N., Volkova E.A., Meshkova R.Ya. The role of IL-33 and IL-1β in the development of persistent allergic rhinitis in children with overweight/obesity. Russian Journal of Allergology. 2022; 19 (4): 472–482. (In Russ.) https://doi.org/10.36691/RJA1575.
25. Cayrol C., Girard J.P. Interleukin-33 (IL-33): A critical review of its biology and the mechanisms involved in its release as a potent extracellular cytokine. Cytokine. 2022; 156: 155891. https://doi.org/10.1016/j.cyto.2022.155891.
26. Hong H., Liao S., Chen F. et al. Role of IL-25, IL-33, and TSLP in triggering united airway diseases toward type 2 inflammation. Allergy. 2020; 75 (11): 2794–2804. https://doi.org/10.1111/all.14526.
27. Chan B.C.L., Lam C.W.K., Tam L.S. et al. IL33: Roles in Allergic Inflammation and Therapeutic Perspectives. Frontiers in immunology. 2019; 10: 364. https://doi.org/10.3389/fimmu.2019.00364.
28. Han J. M., Wu D., Denroche H.C. et al. IL-33 Reverses an Obesity-Induced Deficit in Visceral Adipose Tissue ST2+ T Regulatory Cells and Ameliorates Adipose Tissue Inflammation and Insulin Resistance. Journal of immunology. 2015. 10 (194): 4777–4783. https://doi.org/10.4049/jimmunol.1500020.

29. Lam E.P., Kariyawasam H.H., Rana B.M. IL-25/IL-33-responsive TH2 cells characterize nasal polyps with a default TH17 signature in nasal mucosa. The Journal of allergy and clinical immunology. 2016. 137 (5): 1514–1524. https://doi.org/10.1016/j.jaci.2015.10.019.
30. Matsushita K., Kato Y., Akasaki S. et al. Proallergic cytokines and group 2 innate lymphoid cells in allergic nasal diseases. Allergology international: official journal of the Japanese Society of Allergology. 2015. 64 (3): 235–240. https://doi.org/10.1016/j.alit.2014.12.008.
31. Oliveira M.F., Talvani A., Rocha-Vieira E. IL-33 in obesity: where do we go from here? Inflammation research. 2019; 68 (3): 185–194. https://doi.org/10.1007/s00011-019-01214-2.
32. Glück J., Rymarczyk B., Rogala B. Serum IL-33 but not ST2 level is elevated in intermittent allergic rhinitis and is a marker of the disease severity. Inflammation research: official journal of the European Histamine Research Society. 2012; 61 (6): 547–550. doi: 10.1007/s00011-012-0443-9.
33. Lei Y., Yang H., Zhen L. Obesity is a risk factor for allergic rhinitis in children of Wuhan (China). Asia Pacific Allergy. 2016; 6 (2): 101–104. https://doi.org/10.5415/apallergy.2016.6.2.101.
34. Saadeh D., Salameh P., Caillaud D. et al. High body mass index and allergies in schoolchildren: The french six cities study. BMJ Open Respiratory Research. 2014; 1 (1): e000054. https://doi.org/10.1136/bmjresp-2014-000054.
35. Baumann L.M., Romero K.M., Robinson C.L. et al. Prevalence and risk factors for allergic rhinitis in two resource-limited settings in Peru with disparate degrees of urbanization. Clinical and Experimental Allergy. 2015; 45 (1): 192–199. https://doi.org/10.1111/cea.12379.
36. Lim M.S., Lee C.H., Sim S. et al. Physical Activity, Sedentary Habits, Sleep, and Obesity are Associated with Asthma, Allergic Rhinitis, and Atopic Dermatitis in Korean Adolescents. Yonsei Med J. 2017; 58 (5): 1040–1046. https://doi.org/10.3349/ymj.2017.58.5.1040.
37. Campbell E.A., Qian T., Miller J.M. et al. Identification of temporal condition patterns associated with pediatric obesity incidence using sequence mining and big data. International Journal of Obesity. 2020; 44 (8): 1753–1765. https://doi.org/10.1038/s41366-020-0614-7.
38. Vehapoglu A., Cakın Z.E., Kahraman F.U. et al. Is overweight/obesity a risk factor for atopic allergic disease in prepubertal children? A case–control study. Journal of Pediatric Endocrinology and Metabolism. 2021; 34 (6): 727–732. https://doi.org/10.1515/jpem-2021-0051.
39. Kreißl S., Radon K., Dressel H. et al. Body mass index change and atopic diseases are not always associated in children and adolescents. Ann Allergy Asthma Immunol. 2014; 113 (4): 440–449. https://doi.org/10.1016/j.anai.2014.07.011.
40. Lin M.H., Hsieh C.J., Caffrey J.L. et al. Fetal Growth, Obesity, and Atopic Disorders in Adolescence: Retrospective Birth Cohort Study. Pediatric and Perinatal Epidemiology. 2015; 29 (5): 472–479. https://doi.org/10.1111/ppe.12215.
41. Mai X.M., Almqvist C., Nilsson L., Wickman M. Birth anthropometric measures, body mass index and allergic diseases in a birth cohort study (BAMSE). Archives of Disease in Childhood. 2007; 92 (10): 881–886. https://doi.org/.1136/adc.2006.110692.
42. Chang C.L., Ali G.B., Lodge C.J. et al. Associations between Body Mass Index Trajectories in the first two years of life and Allergic Rhinitis, Eczema and Food Allergy outcomes up to early adulthood. Pediatric Allergy and Immunology. 2022; 33 (3): e13765. https://doi.org/10.1111/pai.13765.
43. Weinmayr G., Forastiere F., Büchele G. et al. ISAAC Phase Two Study Group. Overweight/obesity and respiratory and allergic disease in children: international study of asthma and allergies in childhood (ISAAC) phase two. PLoS One. 2014; 9 (12): e113996. https://doi.org/10.1371/journal.pone.0113996.
44. Kusunoki T., Morimoto T., Nishikomori R. et al. Obesity and the prevalence of allergic diseases in schoolchildren. Pediatric allergy and immunology: official publication of the European Society of Pediatric Allergy and Immunology. 2008; 19 (6): 527–534. https://doi.org/10.1111/j.1399-3038.2007.00686.x.
45. Leung T.F., Kong A.P., Chan I.H. et al. Association between obesity and atopy in Chinese schoolchildren. International Archives of Allergy and Immunology. 2009; 149 (2): 1330–1340. https://doi.org/10.1159/000189196.
46. Skaaby T., Taylor A.E., Thuesen B.H. et al. Estimating the causal effect of body mass index on hay fever, asthma and lung function using Mendelian randomization. Allergy. 2018; 73 (1): 153–164. https://doi.org/10.1111/all.13242.
47. Han Y.Y., Forno E., Gogna M. et al. Obesity and rhinitis in a nationwide study of children and adults in the United States. Journal of Allergy and Clinical Immunology. 2016; 137 (5): 1460–1465. https://doi.org/10.1016/j.jaci.2015.12.1307.
48. Girsh Y.V., Gerasimchik O.A. The role and place of bioimpedance analysis assessment of body composition of children and adolescents with different body mass. Bulletin of Siberian Medicine. 2018; 17 (2): 121–132. (In Russ.)

49. Javed A., Jumean M., Murad M.H. et al. Diagnostic performance of body mass index to identify obesity as defined by body adiposity in children and adolescents: a systematic review and meta-analysis. Pediatric Obesity. 2015; 10 (3): 234–244. doi: 10.1111/ijpo.242.
50. Morąg B., Kozubek P., Gomułka K. Obesity and Selected Allergic and Immunological Diseases-Etiopathogenesis, Course and Management. Nutrients. 2023; 15 (17): 3813. https://doi.org/10.3390/nu15173813.
51. Nikolaev D.V., Rudnev S.G. Bioimpedance analysis: basics of the method, examination protocol and interpretation of results (lecture). Sports medicine: science and practice. 2012; 2: 29–37. (In Russ.)
52. Abdrakhmanova Sh.Z., Aringazina A.M., Adaeva A.A. Methods for school-age children nutritional status assessment: focus on anthropometric parameters. A review. 2023; 6 (25): 207–216. (In Russ.) https://doi.org/10.34689/SH.2023.25.6.024.
53. Li R.L., Wu C.T., Chen S.M. et al. Allergic rhinitis children with obesity are more vulnerable to air pollution: A cross sectional study. Scientific Report. 2023; 13: 3658. https://doi.org/10.1038/s41598-023-30388-3.
54. Bekezin V.V., Koroleva A.E., Sazonekova L.V., Volkova E.V., Meshkova R.Ya. Allergic rhinitis in children with overweight/obesity: features of sensitization. Allergology and immunology in pediatrics. 2020; 63 (4): 29–35. (In Russ.) https://doi.org/10.24411/2500-1175-2020-1001.
55. Park D.Y., Kim Y.S., Kim J.H. et al. Association of body mass index and other factors with histamine skin reactivity in adults with allergic nasal symptoms. The American Journal of Rhinology & Allergy. 2015; 29 (6): 160–163. https://doi.org/10.2500/ajra.2015.29.4233.
56. Ramalho R., Almeida J., Beltrao M. et al. Neurogenic inflammation in allergen-challenged obese mice: A missing link in the obesity-asthma association? Experimental Lung Research. 2012; 38: 316–324. https://doi.org/10.3109/01902148.2012.699589.
57. Furukawa S., Asano K., Kobayashi H. Suppressive Activity of Adiponectin on the Development of Allergic Rhinitis in Mice. In Vivo. 2019; 33 (1): 93–98. https://doi.org/10.21873/invivo.11444.
58. Park J.H., Yoo E., Seo M.W. et al. Association between Physical Activity and Respiratory Diseases in Adolescents: An Age- and Gender-Matched Study. International journal of environmental research and public health. 2021; 18 (4): 1397. https://doi.org/10.1016/j.jshs.2020.09.009.
59. de Sá Pittondo M., Migueis D.P., Fujita R.R., et al. Effect of Body Weight on Response to Nasal Glucocorticoid Treatment in Allergic Rhinitis. Indian Journal of Otolaryngology and Head and Neck Surgery. 2024; 76 (1): 1002–1009. https://doi.org/10.1007/s12070-023-04344-6.

ЛИТЕРАТУРА

1. Passali D., Cingi C., Staffa P. et al. The International Study of the Allergic Rhinitis Survey: outcomes from 4 geographical regions. Asia Pacific Allergy. 2018; 8 (1): 1–15. https://doi.org/10.5415/apallergy.2018.8.e7.
2. Bousquet J., Anto J.M., Bachert C. et al. Allergic rhinitis. Nature reviews. Disease primers. 2020; 6 (1): 95. https://doi.org/10.1038/s41572-020-00227-0.
3. Zhou J., Luo F., Han Y. et al. Obesity/overweight and risk of allergic rhinitis: A meta-analysis of observational studies. Allergy: European Journal of Allergy and Clinical Immunology. 2020; 75 (5): 1272–1275. https://doi.org/10.1111/all.14143.
4. Kelishadi R., Roufarshbaf M., Soheili S. et al. Association of Childhood Obesity and the Immune System: A Systematic Review of Reviews. Childhood Obesity. 2017; 13 (4): 332–346. https://doi.org/10.1089/chi.2016.0176.
5. Мигачева Н.Б., Скворцова О.В., Каганова Т.И., Гинзбург А.С. Парадоксы иммунного ответа при ожирении. Эффективная фармакотерапия. 2023; 19 (28): 30–36.
6. Recinella L., Orlando G., Ferrante C. et al. Adipokines: New Potential Therapeutic Target for Obesity and Metabolic, Rheumatic and Cardiovascular Diseases. Frontiers in physiology. 2020; 11: 1–32. https://doi.org/10.3389/fphys.2020.578966.
7. Umano G.R., Pistone C., Tondina E. et al. Pediatric Obesity and the Immune System. Frontiers in Pediatrics. 2019; 7: 1–9. https://doi.org/10.3389/fped.2019.00487.
8. Yeo B.S.Y., Guan E.J., Ng K. et al. Association of Abnormal Body Weight and Allergic Rhinitis-A Systematic Review and Meta-Analysis. Clinical and Experimental Allergy. 2025; 55 (2): 142–165. https://doi.org/10.1111/cea.14604.
9. Tajima H., Pawankar R. Obesity and adiposity indicators in asthma and allergic rhinitis in children. Current opinion in allergy and clinical immunology. 2019; 19 (1): 7–11. https://doi.org/10.1097/ACI.0000000000000504.
10. Stefani C., Pecoraro L., Flodmark C.E. et al. Allergic Diseases and Childhood Obesity: A Detrimental Link? Biomedicines. 2023; 11: 2061. https://doi.org/10.3390/biomedicines11072061.
11. Wang X., Shen Y., Ke X. et al. Role of leptin/ILC2 axis in allergic rhinitis in obese children. International journal of pediatric otorhinolaryngology. 2022; 157. https://doi.org/10.1016/j.ijporl.2022.111127.

12. Wang X., Hu G., Shen Y. et al. Leptin mediated activation of group 2 innate lymphocytes aggravates the pathogenesis of allergic rhinitis in obese adults. 2022; 38 (12): 1118–1124.
13. Liu W., Zeng Q., Zhou L. et al. Association of leptin with disease severity and inflammation indicators in Chinese obese children with allergic rhinitis. Pediatric allergy and immunology. 2018; 29 (2): 186–193. https://doi.org/10.1111/pai.12856.
14. Hsueh K.C., Lin Y.J., Lin H.C. et al. Serum leptin and adiponectin levels correlate with severity of allergic rhinitis. Pediatric allergy and immunology: official publication of the European Society of Pediatric Allergy and Immunology. 2010; 21 (1): 155–159. https://doi.org/10.1111/j.1399-3038.2009.00878.x.
15. Unal M., Eskandari G., Muşlu N. et al. Serum leptin levels in patients with allergic rhinitis. Otolaryngology-head and neck surgery: official journal of American Academy of Otolaryngology-Head and Neck Surgery. 2006; 134 (2): 331–333. https://doi.org/10.1016/j.otohns.2005.11.021.
16. Zeng Q., Luo X., Han M. et al. Leptin/Osteopontin Axis Regulated Type 2T Helper Cell Response in Allergic Rhinitis with Obesity. Ebiomedicine. 2018; 32: 43–49. https://doi.org/10.1016/j.ebiom.2018.05.037.
17. Liu W., Zeng Q., Chen Y. et al. Role of leptin/osteopontin axis in the function of eosinophils in allergic rhinitis with obesity. Mediators of Inflammation. 2018: 1–10. https://doi.org/10.1155/2018/9138904.
18. Королева А.Е., Бекезин В.В., Мешкова Р.Я., Демина Е.Г., Цветная И.Н. Фенотипические маркеры аллергического ринита у детей на фоне избыточной массы тела и ожирения. Российский вестник перинатологии и педиатрии. 2023; 68 (4): 351.
19. Kalpaklıoglu A.F., Baccioglu A., Yalim S.A. Does serum leptin levels differ between patients with rhinitis of allergic versus nonallergic etiology? The clinical respiratory journal. 2021; 15 (12): 1352–1358. https://doi.org/10.1111/crj.13440.
20. Shore S.A., Terry R.D., Flynt L. et al. Adiponectin attenuates allergen-induced airway inflammation and hyperresponsiveness in mice. The Journal of allergy and clinical immunology. 2006; 118 (2): 389–395. https://doi.org/10.1016/j.jaci.2006.04.021.
21. Wang H.R., Wei S.Z., Song X.Y. et al. IL-1β and Allergy: Focusing on Its Role in Allergic Rhinitis. Mediators of Inflammation. 2023: 1265449. https://doi.org/10.1155/2023/1265449.
22. Han M.W., Kim S.H., Oh I. et al. Serum IL-1β can be a biomarker in children with severe persistent allergic rhinitis. Allergy Asthma Clin Immunol. 2019; 18 (15): 58. https://doi.org/10.1186/s13223-019-0368-8.
23. Han M.W., Kim S.H., Oh I. et al. Obesity Can Contribute to Severe Persistent Allergic Rhinitis in Children through Leptin and Interleukin-1β. International Archives of Allergy and Immunology. 2021; 182 (6): 546–555. https://doi.org/10.1159/000512920.
24. Королева А.Е., Бекезин В.В., Сергеева И.Н., Волкова Е.А., Мешкова Р.Я. Роль IL-33 и IL-1β в развитии персистирующего аллергического ринита у детей с избыточной массой тела / ожирением. Российский аллергологический журнал. 2022; 19 (4): 472–482. https://doi.org/10.36691/RJA1575.
25. Cayrol C., Girard J.P. Interleukin-33 (IL-33): A critical review of its biology and the mechanisms involved in its release as a potent extracellular cytokine. Cytokine. 2022; 156: 155891. https://doi.org/10.1016/j.cyto.2022.155891.
26. Hong H., Liao S., Chen F. et al. Role of IL-25, IL-33, and TSLP in triggering united airway diseases toward type 2 inflammation. Allergy. 2020; 75 (11): 2794–2804. https://doi.org/10.1111/all.14526.
27. Chan B.C.L., Lam C.W.K., Tam L.S. et al. IL33: Roles in Allergic Inflammation and Therapeutic Perspectives. Frontiers in immunology. 2019; 10: 364. https://doi.org/10.3389/fimmu.2019.00364.
28. Han J. M., Wu D., Denroche H.C. et al. IL-33 Reverses an Obesity-Induced Deficit in Visceral Adipose Tissue ST2+ T Regulatory Cells and Ameliorates Adipose Tissue Inflammation and Insulin Resistance. Journal of immunology. 2015; 10 (194): 4777–4783. https://doi.org/10.4049/jimmunol.1500020.
29. Lam E.P., Kariyawasam H.H., Rana B.M. IL-25/IL-33-responsive TH2 cells characterize nasal polyps with a default TH17 signature in nasal mucosa. The Journal of allergy and clinical immunology. 2016. 137 (5): 1514–1524. https://doi.org/10.1016/j.jaci.2015.10.019.
30. Matsushita K., Kato Y., Akasaki S. et al. Proallergic cytokines and group 2 innate lymphoid cells in allergic nasal diseases. Allergology international: official journal of the Japanese Society of Allergology. 2015; 64 (3): 235–240. https://doi.org/10.1016/j.alit.2014.12.008.
31. Oliveira M.F., Talvani A., Rocha-Vieira E. IL-33 in obesity: where do we go from here? Inflammation research. 2019; 68 (3): 185–194. https://doi.org/10.1007/s00011-019-01214-2.
32. Glück J., Rymarczyk B., Rogala B. Serum IL-33 but not ST2 level is elevated in intermittent allergic rhinitis and is a marker of the disease severity. Inflammation research: official journal of the European Histamine Research Society. 2012; 61 (6): 547–550. doi: 10.1007/s00011-012-0443-9.

33. Lei Y., Yang H., Zhen L. Obesity is a risk factor for allergic rhinitis in children of Wuhan (China). Asia Pacific Allergy. 2016; 6 (2): 101–104. https://doi.org/10.5415/apallergy.2016.6.2.101.
34. Saadeh D., Salameh P., Caillaud D. et al. High body mass index and allergies in schoolchildren: The french six cities study. BMJ Open Respiratory Research. 2014; 1 (1): e000054. https://doi.org/10.1136/bmjresp-2014-000054.
35. Baumann L.M., Romero K.M., Robinson C.L. et al. Prevalence and risk factors for allergic rhinitis in two resource-limited settings in Peru with disparate degrees of urbanization. Clinical and Experimental Allergy. 2015; 45 (1): 192–199. https://doi.org/10.1111/cea.12379.
36. Lim M.S., Lee C.H., Sim S. et al. Physical Activity, Sedentary Habits, Sleep, and Obesity are Associated with Asthma, Allergic Rhinitis, and Atopic Dermatitis in Korean Adolescents. Yonsei Med J. 2017; 58 (5): 1040–1046. https://doi.org/10.3349/ymj.2017.58.5.1040.
37. Campbell E.A., Qian T., Miller J.M. et al. Identification of temporal condition patterns associated with pediatric obesity incidence using sequence mining and big data. International Journal of Obesity. 2020; 44 (8): 1753–1765. https://doi.org/10.1038/s41366-020-0614-7.
38. Vehapoglu A., Cakın Z.E., Kahraman F.U. et al. Is overweight/obesity a risk factor for atopic allergic disease in prepubertal children? A case–control study. Journal of Pediatric Endocrinology and Metabolism. 2021; 34 (6): 727–732. https://doi.org/10.1515/jpem-2021-0051.
39. Kreißl S., Radon K., Dressel H. et al. Body mass index change and atopic diseases are not always associated in children and adolescents. Ann Allergy Asthma Immunol. 2014; 113 (4): 440–449. https://doi.org/10.1016/j.anai.2014.07.011.
40. Lin M.H., Hsieh C.J., Caffrey J.L. et al. Fetal Growth, Obesity, and Atopic Disorders in Adolescence: Retrospective Birth Cohort Study. Pediatric and Perinatal Epidemiology. 2015; 29 (5): 472–479. https://doi.org/10.1111/ppe.12215.
41. Mai X.M., Almqvist C., Nilsson L., Wickman M. Birth anthropometric measures, body mass index and allergic diseases in a birth cohort study (BAMSE). Archives of Disease in Childhood. 2007; 92 (10): 881–886. https://doi.org/.1136/adc.2006.110692.
42. Chang C.L., Ali G.B., Lodge C.J. et al. Associations between Body Mass Index Trajectories in the first two years of life and Allergic Rhinitis, Eczema and Food Allergy outcomes up to early adulthood. Pediatric Allergy and Immunology. 2022; 33 (3): e13765. https://doi.org/10.1111/pai.13765.
43. Weinmayr G., Forastiere F., Büchele G. et al. ISAAC Phase Two Study Group. Overweight/obesity and respiratory and allergic disease in children: international study of asthma and allergies in childhood (ISAAC) phase two. PLoS One. 2014; 9 (12): e113996. https://doi.org/10.1371/journal.pone.0113996.
44. Kusunoki T., Morimoto T., Nishikomori R. et al. Obesity and the prevalence of allergic diseases in schoolchildren. Pediatric allergy and immunology: official publication of the European Society of Pediatric Allergy and Immunology. 2008; 19 (6): 527–534. https://doi.org/10.1111/j.1399-3038.2007.00686.x.
45. Leung T.F., Kong A.P., Chan I.H. et al. Association between obesity and atopy in Chinese schoolchildren. International Archives of Allergy and Immunology. 2009; 149 (2): 1330–1340. https://doi.org/10.1159/000189196.
46. Skaaby T., Taylor A.E., Thuesen B.H. et al. Estimating the causal effect of body mass index on hay fever, asthma and lung function using Mendelian randomization. Allergy. 2018; 73 (1): 153–164. https://doi.org/10.1111/all.13242.
47. Han Y.Y., Forno E., Gogna M. et al. Obesity and rhinitis in a nationwide study of children and adults in the United States. Journal of Allergy and Clinical Immunology. 2016; 137 (5): 1460–1465. https://doi.org/10.1016/j.jaci.2015.12.1307.
48. Гирш Я.В., Герасимчик О.А. Роль и место биоимпедансного анализа в оценке состава тела детей и подростков с различной массой тела. Бюллетень сибирской медицины. 2018; 17 (2): 121–132.
49. Javed A., Jumean M., Murad M.H. et al. Diagnostic performance of body mass index to identify obesity as defined by body adiposity in children and adolescents: a systematic review and meta-analysis. Pediatric Obesity. 2015; 10 (3): 234–244. doi: 10.1111/ijpo.242.
50. Morąg B., Kozubek P., Gomułka K. Obesity and Selected Allergic and Immunological Diseases-Etiopathogenesis, Course and Management. Nutrients. 2023; 15 (17): 3813. https://doi.org/10.3390/nu15173813.
51. Николаев Д.В., Руднев С.Г. Биоимпедансный анализ: основы метода, протокол обследования и интерпретация результатов (лекция). Спортивная медицина: наука и практика. 2012; 2: 29–37.
52. Абдрахманова Ш.З., Арингазина А.М., Адаева А.А. Методы оценки состояния питания детей школьного возраста: фокус на антропометрических параметрах. Обзор литературы. Наука и здравоохранение. 2023; 6 (25): 207–216. https://doi.org/10.34689/SH.2023.25.6.024.
53. Li R.L., Wu C.T., Chen S.M. et al. Allergic rhinitis children with obesity are more vulnerable to air pollution: A cross sectional study. Scientific Report. 2023; 13: 3658. https://doi.org/10.1038/s41598-023-30388-3.

54. Бекезин В.В., Королева А.Е., Сазоненкова Л.В., Волкова Е.В., Мешкова Р.Я. Аллергический ринит у детей с избыточной массой тела / ожирением: особенности сенсибилизации. Аллергология и иммунология в педиатрии. 2020; 63 (4): 29–35. https://doi.org/10.24411/2500-1175-2020-1001.
55. Park D.Y., Kim Y.S., Kim J.H. et al. Association of body mass index and other factors with histamine skin reactivity in adults with allergic nasal symptoms. The American Journal of Rhinology & Allergy. 2015; 29 (6): 160–163. https://doi.org/10.2500/ajra.2015.29.4233.
56. Ramalho R., Almeida J., Beltrao M. et al. Neurogenic inflammation in allergen-challenged obese mice: A missing link in the obesity-asthma association? Experimental Lung Research. 2012; 38: 316–324. https://doi.org/10.3109/01902148.2012.699589.
57. Furukawa S., Asano K., Kobayashi H. Suppressive Activity of Adiponectin on the Development of Allergic Rhinitis in Mice. In Vivo. 2019; 33 (1): 93–98. https://doi.org/10.21873/invivo.11444.
58. Park J.H., Yoo E., Seo M.W. et al. Association between Physical Activity and Respiratory Diseases in Adolescents: An Age- and Gender-Matched Study. International journal of environmental research and public health. 2021; 18 (4): 1397. https://doi.org/10.1016/j.jshs.2020.09.009.
59. de Sá Pittondo M., Migueis D.P., Fujita R.R., et al. Effect of Body Weight on Response to Nasal Glucocorticoid Treatment in Allergic Rhinitis. Indian Journal of Otolaryngology and Head and Neck Surgery. 2024; 76 (1): 1002–1009. https://doi.org/10.1007/s12070-023-04344-6.

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Anna E. Koroleva — 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.

Vladimir V. Bekezin — conceptualization.

Raisa Y. Meshkova — conceptualization, writing — review & editing — preparation, creation and/or presentation of the published work by those from the original research group, specifically critical review.

ВКЛАД АВТОРОВ В РАБОТУ

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