

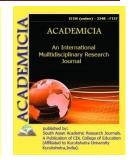
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METHODS OF DIETOTHERAPY AND DIETODIAGNOSTICS FOR DIFFERENT TYPES OF FOOD INTOLERANCE IN CHILDREN

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ABSTRACT

Food intolerance is one of the most pressing problems of modern clinical and preventive medicine. The purpose of this article is to familiarize pediatricians with the main types of food intolerances, their diagnosis and treatment using simple elimination diets. When analyzing the results of the study after the course of diet therapy, positive dynamics was noted, which was confirmed by a significant increase in anthropometric data (weight and BMI).

KEYWORDS: *Diet Therapy, Dietetics, Children, Food Intolerances*

INTRODUCTION

It is difficult to find a patient who, at least once in his life, has not encountered the manifestations of certain reactions of the body to food. For the first time, such reactions are usually noted in childhood, although they can occur throughout a person's life. The first indications of unusual reactions to food are known since the time of Hippocrates (460-370 BC), who described adverse reactions to food in the form of gastrointestinal and skin symptoms when

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drinking cow's milk. Later, in the XVI-XVII centuries, anaphylactic reactions to eggs and fish were described, but in the XX century a qualitative change occurred in the composition of the human diet, and "unusual" reactions to food became ubiquitous, which served as a powerful impetus to the study of the phenomena of food intolerance (PN) and food allergy (PA) [1,3,4].

Food intolerance is a common cause of long-term stool disorders in infants [2,5,6,7,8]. An analysis of the case histories of children with prolonged diarrhea shows that in their treatment, the main efforts were directed at combating pathogenic flora and dysbiosis, while dietary correction, if carried out, was timid and inconsistent [9,10,18].

Symptoms of allergies and food intolerances are usually very similar - itching of the skin and mucous membranes, rash, abdominal discomfort, stool disturbances - but the mechanisms are different. An allergic reaction to a product is the body's immune response to an irritant; at the same time, the immune system perceives food proteins as foreign and develops protective antibodies - immunoglobulins E (IgE). The latter provoke an allergic reaction - most often to milk derivatives, eggs, fish and seafood, wheat protein, nuts or soy. As soon as contact with the allergen ceases, the immune system calms down and the allergy symptoms subside [11,13,17,21,25].

In the case of food intolerance (also called "pseudo-allergy"), the immune system does not overreact. Sometimes they say that this condition is associated with the production of type G immunoglobulins (IgG), but Marina Vershinina notes that, unlike IgE, markers of allergy, class G antibodies are the result of a normal reaction of the immune system, that is, an indicator that the body has become acquainted with that or any other food product and is not going to react to it. The doctor emphasizes that laboratory tests for the determination of IgG are not cheap, but the detection of such antibodies in itself does not allow drawing conclusions about the sensitivity to some products [14,16,22,23,24].

If a small amount or even one smell of an allergen is enough for the appearance of allergy symptoms, then food intolerance is more complicated. In the case of pseudo-allergy, the reaction of the body is often delayed (sometimes by several days), and the manifestations are blurred. For example, abdominal pain can be accompanied by fatigue or nasal congestion, which can easily be attributed to overwork or a cold. At the same time, according to doctor Mikhail Gavrilov, food intolerance occurs in adults much more often than food allergies. At risk are those who have metabolic problems or diseases of the gastrointestinal tract, as well as smokers and people who exercise little [12,15,19,20].

According to Marina Vershinina, a separate disease "food intolerance" does not exist at all - but there are many diseases and conditions, against the background of which intolerance of certain substances in food may appear. This may be due to malfunctions in the production of enzymes that digest this or that component (for example, with a lack of the lactase enzyme, the body does not process lactose, milk sugar). The reasons can be inflammation of the intestinal mucosa during infections, and autoimmune processes, and the toxic effect of some products or drugs. Hypersensitivity to chemical food additives - preservatives, dyes, emulsifiers - is also not uncommon [25,26,27,28,29].

Most often, a failure occurs due to the fact that certain enzymes are not produced enough. Usually in children, this is due to the immaturity of the gastrointestinal tract, and in adults, for



example, with inflammation of the pancreas. Lactose intolerance usually begins in adults and is considered a normal aging process. Whole milk is a staple of nutrition at an early age, but over time, the production of an enzyme that breaks down milk sugar decreases. According to Mikhail Gavrilov, almost a third of people over twenty-five years old notice a worsening of their condition after a glass of whole milk, although they did not have such a problem before. Fructose intolerance develops due to malabsorption - while the work of the so-called fructose carriers, that is, the molecules responsible for the passage of the substance through the intestinal wall, breaks down. Its symptoms - nausea, vomiting, flatulence, diarrhea - resemble poisoning [30, 33, 34, 38, 40].

According to allergist-immunologist Ekaterina Koroteeva, it is not an easy task to detect and cure food intolerance: there are no reliable clinical and laboratory criteria for pseudoallergy. Therefore, when diagnosing, doctors use the exclusion method, first brushing aside food allergies and celiac disease (gluten intolerance, which is based on genetic and autoimmune factors). Stool analysis and a breath hydrogen test can help confirm lactose or fructose intolerance - it detects disorders in the breakdown and absorption of carbohydrates, which often lead to bloating and diarrhea; but in this case the diagnosis will be only preliminary [31,32,35,41,43].

Your doctor will advise you to keep a food diary, recording the foods you eat during the day and how you feel after breakfast, lunch and dinner. The next step is an elimination diet. The specialist removes one "suspicious" component from the diet; If, after six weeks of the diet, the condition improves, then the forbidden food is the culprit of the poor state of health. After that, such a product can be introduced into the diet, but in small portions and with awareness of the consequences. If there is still no improvement, then you need to try another option [36,37,38,39,42,44].

The purpose of this article is to familiarize pediatricians with the main types of food intolerances, their diagnosis and treatment using simple elimination diets. Lactose (milk sugar) intolerance - the most common form of prolonged diarrhea - is associated with a decrease in the activity of lactase, which is parietally in the brush border of the enterocytes of the small intestine. As a result, a significant part of the non-hydrolyzed lactose enters the large intestine, where it undergoes microbial fermentation with significant gas formation, a decrease in fecal pH to 4.0-5.0 and a delay in water absorption.

Sour, watery stools are a reliable sign of lactase deficiency. In most cases, a decrease in lactase activity occurs under the influence of intestinal infections (if bacterial, rotavirus, etc.), which is manifested by the lack of normalization of the stool against the background of normal feeding.

The better tolerance of breast milk compared to cow's milk may be due to the predominance of alactose in it, while cow's milk mainly contains B-lactose. Slower passage of breast milk through the intestine can also promote more complete breakdown of lactose while partially retaining lactase activity.

Much less common are primary forms of a hereditary nature, in which stool disorders are not associated with a previous infection, but are caused by congenital lactase deficiency.

Intolerance to cow's milk proteins (more often B-lactoglobulin), the second most common type of intolerance, is manifested by stubborn, watery stools, often mixed with blood, the rapid development of hypotrophy, and sometimes an allergic rash. Children are usually reluctant to eat



formula milk, regurgitate. In most cases, the deterioration of the child's condition is associated with the introduction of dairy products into the diet.

A fairly common form of intolerance is celiac disease (celiac disease), in which contact of the mucous membrane of the small intestine with protein (gluten) leads to its atrophy with a sharp malabsorption. Celiac disease is characterized by abundant (polyfecal-daily stool weight reaches 200-300g), fatty, often frothy stools, malnutrition (body weight deficit 20-40%), an increase in abdominal volume. Gluten is found in wheat, rye, barley, and oats. Children tolerate rice, buckwheat, corn well. Many children with celiac disease are intolerant to milk protein and tend to be lactose intolerant. On the other hand, gluten intolerance (usually temporary) is quite common with milk protein intolerance.

Congenital sugar and starch intolerance is much less common. It is associated with a defect in the corresponding disaccharidases, sucrase, isomaltase and amylase. In children with prolonged diarrhea, however, one has to reckon with the possibility of a secondary decrease in the activity of these enzymes and correct the diet.

Sometimes there is an intolerance to many proteins (soy, various types of meat), manifested by diarrhea. It is associated with increased permeability of intestinal membranes, but the role of an immunological factor cannot be excluded, since atopic dermatitis is often observed in these children.

Disorders of abdominal digestion in children with food intolerance are common, but they usually do not reach a significant degree, while the violation of membrane digestion can be pronounced. Enzyme replacement therapy without diet therapy usually does not work, these drugs have to be used to improve the digestion of protein and fat.

The role of the microbial factor in children with food intolerances is usually secondary; against the background of prolonged diarrhea, as a rule, dysbiosis develops, and in some children, pathogenic flora is also released.

Dysbacteriosis is primarily associated with a violation of the habitat of intestinal microorganisms, therefore, restoration of normal intestinal function plays a leading role in its treatment.

Diet therapy, especially with the use of fermented milk mixtures, is the most effective way to normalize the intestinal flora. Antibiotics for dysbiosis are contraindicated, the use of eubiotics (bifidumbacterin, colibacterin) often leads to deterioration of the stool. Based on the experience of treating more than 150 patients with dysbiosis, we were convinced that adequate dietary correction, by normalizing the stool, also ensures the normalization of flora, including after long-term unsuccessful therapy with anti-eubiotics (2,6).

Step-by-step dietary diagnostics. When investigating the causes of prolonged diarrhea, it is important to establish the underlying cause. The complexity of diagnosis is that the use of special tests in the presence of diarrhea in a child is usually not possible. We have developed a simple method of "diet diagnosis" that allows you to simultaneously correct the child's diet.

With prolonged (more than 3 weeks) diarrhea with watery stools, low pH (less than 5.5) and gas formation, the diagnosis of lactase deficiency is most likely. In children who are on mixed and artificial feeding, milk and any form are excluded from the diet, dairy products are replaced by a



low-lactose product. For this purpose, children who do not receive breast milk have 3-day kefir (1.1-1.65 lactose), but other low-lactose products can be used instead.

If babies are receiving breast milk, do not immediately switch to low-lactose formulas. Often, with hypolactasia, a good effect is given by replacing half of breast milk with these mixtures at each feeding. This ensures a decrease in the total concentration of lactose and allows you to maintain breastfeeding. In a number of patients with alactasia, stool normalization occurs only after the complete replacement of breast milk with a lactose-free mixture.

Stool normalization with the use of 3-day kefir gives grounds to diagnose lactase deficiency. This diagnosis can be confirmed either with the help of a load of lactose (1 g per 1 kg of body weight), or by provocation with milk (20 ml per 1 kg of body weight): the appearance of acidic watery stools, bloating indicate a decrease in lactase activity. Lactase deficiency is also evidenced by a flat glycemic curve after loading with lactose (an increase in glucose levels after 30-90 minutes than by 20 mg / 1). This test is carried out only against the background of persistently normal stool; in children with malnutrition, it is advisable to postpone the test until body weight is restored.

Snowing of lactase activity in secondary forms of lactase deficiency is observed from several weeks to many months: a sample with milk allows, if necessary, to track the dynamics of its recovery. The method for diagnosing lactase deficiency is a chromatographic study of the excretion of sugars in the feces.

With the preservation of loose stools, no increase in body weight, despite the use of low-lactose mixtures, the diagnosis of milk protein intolerance is very likely. In these cases, the next step in diet therapy is the complete elimination of dairy products. At the beginning of treatment, it is also advisable to exclude foods containing gluten and beef. Soy-based formulas are considered the best milk replacer, but in our experience diarrhea can persist with these formulas.

If on a dairy-free diet, the stool usually returns to normal after 2-5 days, then the diagnosis of cow's milk protein intolerance becomes obvious. In a number of children with this form, it is also relatively easy to typify this form with the help of elimination diets. Among the more rare diseases can be called exudative enteropathy in a child with cirrhosis of the liver and exocrine pancreatic insufficiency (Schwachman's syndrome), in children with Crohn's disease. Persistent diarrhea is observed in children with combined forms of immunodeficiency; in these cases, the results of the study of three classes of immunoglobulins are decisive for the diagnosis. We observe persistent diarrhea with hypokalemia in a 2-year-old girl with a tumor of the retroperitoneal space. In making the diagnosis in these cases, in addition to special studies, the lack of effect from elimination diets helped.

A syndrome reminiscent of celiac disease is characteristic of the intestinal form of cystic fibrosis, for which an increase in sweat chloride and usually a normal result of a test with D-xylose is typical: a gluten-free table does not improve stool in these patients.

Diet therapy. As low-lactose milk mixtures, along with 3-day kefir, you can use a low-lactose mixture with malt extract, which is not inferior in efficiency to the Nutrilon premium mixture (lactose-free) produced in Uzbekistan. Instead of kefir, of course, you can use any other lactic acid product (yogurt, acidophilus, etc.) with a correspondingly extended production time.



Children with lactase deficiency, along with the above mixtures, receive dairy-free porridge, cottage cheese, washed from whey. Meat, vegetables, fruits are introduced as usual. Additional prescription of enzyme preparations or eubiotics, as a rule, is not required.

To feed children with cow's milk intolerance, cereals on the water (rice, buckwheat), mashed potatoes, cabbage are used. Various meats are used as a source of protein (lean pork, lamb, turkey); useful horse meat (fresh or canned), which we observed good tolerance even in children of the 2nd month of life. The amount of meat increases from 5-10 to 100 g per day for 7-10 days. The diet is enriched with lard or vegetable oil. The total amount of food increases gradually, so that 70% of the calorie content is reached within 5-7 days. Due to the possibility of disaccharidase deficiency, it is good to include glucose or fructose in the diet. Of enzyme preparations, it may be necessary to prescribe oraza. Pancreatin and preparations containing it (cryon, festal, panzinorm, etc.) can support the phenomena of intolerance. It is advisable to expand the diet no earlier than 1 month after stool normalization.

In children with persistent diarrhea against the background of parenteral nutrition, small amounts of food are administered from 2 to 3 hectares of the day: 20-30 g per intake of a 5% solution of monosaccharides, rice broth or liquid rice porridge, which children usually tolerate well, without deteriorating stool. It is best to start treatment with the introduction of blood plasma inside, 30-50 ml per day. If you are intolerant of starch, instead of porridge, you can try to give mashed cabbage (cauliflower, Brussels sprouts, white cabbage), zucchini, bananas. In the future, from the 7-10th day, the amount of food is brought to normal, vegetable oil is gradually added to the porridge or puree, the type of meat is individually selected.

Parenteral nutrition is a very demanding procedure; the introduction of highly osmolar fluids (usually it is necessary to inject a 15-20% glucose solution, alvezin, lipofundin) is fraught with endothelial injury and the formation of septic thrombi. Therefore, it should be resorted to only in extreme cases and strive for the fastest possible transition to enteral nutrition. Some children have to leave a minimal set of foods (for example, rice porridge, vegetable oil, horse meat, glucose or fructose) for many months; in any case, when expanding the diet, it is necessary to introduce one new product at a time under the control of the stool character.

When confirming the diagnosis of celiac disease, the diet should be strictly gluten-free, since even small amounts of gluten interfere with the restoration of the function of the intestinal mucosa. Rice, buckwheat and corn are widely used. It is necessary to ensure that children do not receive gluten with ready-made products (boiled sausage, canned food in tomato and mustache, to which flour is often added). Many children with celiac disease have reduced lactase activity, which is often accompanied by intolerance to milk proteins. At the beginning of treatment, it is recommended to exclude dairy products. The diet includes different types of meat, fruits, vegetables, fats. Due to poor absorption at the beginning of treatment, the amount of fat can be reduced, it is advisable to prescribe lipase preparations (oraza, pancreatin, panzinorm).

Additional therapeutic effects. Rapid stool normalization and good weight gain in most children, against the background of adequate dietary therapy, make other types of treatment superfluous. Nevertheless, given the limited set of products in the diet of some children, it is necessary to additionally introduce vitamins, they are mandatory for parenteral nutrition.



Above it was said about the use of enzyme preparations to improve intestinal digestion. Abomin, hydrochloric acid with pepsin is indicated for children who eat large amounts of meat. Children on a dairy-free diet should be given calcium salts. In patients with celiac disease, hypocalcemic convulsions may develop during treatment, which requires parenteral calcium administration, as well as the appointment of vitamin D, which improves calcium absorption in the intestine. In the acute phase of the disease, with large fluid losses, it is necessary to correct the water-salt balance and the balance of acids and bases.

CONCLUSIONS:

1. The developed method of diet therapy and dietary diagnostics is more perfect and is shown for long-term use without prejudice to the physical and mental development of infants.

2. When analyzing the results of the study after the course of diet therapy, positive dynamics was noted, which was confirmed by a significant increase in anthropometric data (weight and BMI).

REFERENCES:

- 1. Belmer S.V., Mukhina Yu.G., Chubarova A.I., Geraskina V.P., Gasilina T.V. Lactose intolerance in children and adults // Attending physician. -2005. -No. 1.
- **2.** Drobyshevskaya V.A. The method of nutritional therapy for alimentary obesity. / Problems of health and ecology. 2005.S. 87-90.
- **3.** Zaykov S.V., Bogomolov A.E. Food intolerance and food allergy // Alergology. Look around. Ukraine, 2013.S. 58-60.
- **4.** Zaikov S.V., Bogomolov A.E. Modern approaches to the treatment of food allergies // Alergology. Practical advice ii. Ukraine, 2013.S. 16-18.
- 5. Therapeutic nutrition // Great Russian encyclopedia. Volume 26. -M., 2014 .-- S. 284.
- **6.** Gurvich M. Big encyclopedia of diet therapy (Medical encyclopedia). M .: Eksmo, 2008.-768 p.
- 7. Pokrovsky AA Conversations about nutrition. 3rd ed. -M .: Economics, 1986.-367 p.
- **8.** Kozlov AI Lactase deficiency (primary hypolactasia) in various groups of the population of Eurasia: Dissertation for the degree of Doctor of Biological Sciences: M., 2004. 200 p.
- **9.** Mukhina Yu.G., Chubarova A.I., Geraskina V.P. Modern aspects of the problem of lactase deficiency in young children. Vopr. children dietitian. 2003; 1 (1): 50-6.
- **10.** Mukhina Yu.G., Shumilov P.V., Dubrovskaya M.I. et al. Modern approaches to the diagnosis and treatment of disaccharidase deficiency in children. Difficult. a patient. 2006; 9 (4): 12-6.
- **11.** Sheybak M.P. Lack of intestinal lactase in children (lactose intolerance). Russian Bulletin of Perinatology and Pediatrics. 1995; 4: 45-8.
- **12.** Kornienko E.A., Mitrofanova N.I., Larchenkova L.V. Lactase deficiency in young children. Vopr. modern pediatrician. 2006; 5 (4): 82-6.
- Heyman MB. Lactose Intolerance in Infants, Children, and Adolescents. Pediatrics 2006; 118 (3): 1279–86.

ISSN: 2249-7137 Vol. 11, Issue 5, May 2021

ACADEMICIA

- Impact Factor: SJIF 2021 = 7.492
- 14. Carroccio, Montalto, Cavera, Notarbatolo. Lactose Intolerance and Self-Reported Milk Intolerance: Relationship with Lactose Maldigestion and Nutrient Intake // Journal of the American College of Nutrition: journal. -1998. -Vol. 17. -P. 631-636.
- 15. McGee, Harold. Milk after infancy: dealing with lactose // On Food and Cooking (Revised Edition). -Scribner, 2004. – pp. 14–15.
- 16. Sicherer S.H., Sampson H.A. Food allergy: recent advances in pathophysiology and treatment. Annu Rev Med. 2009; 60: 261-277.
- 17. Sicherer S.H. Epidemiology of Food Allergy. J Allergy ClinImmunol. 2010.
- 18. Sicherer S.H., MunozFurlong A., Godbold J.H., Sampson H.A. US prevalence of selfreported peanut, tree nut, and sesame allergy: 11 year followup. J Allergy ClinImmunol. 2010; 125 (6): 1322-1326.
- **19.** Gupta R., Sheikh A., Strachan D.P., Anderson H.R. Time trends in allergic disorders in the UK. Thorax. 2007; 62 (1): 91-96.
- 20. Decker W.W., Campbell R.L., Manivannan V. et al. The etiology and incidence of anaphylaxis in Rochester, Minnesota: a report from the Rochester Epidemiology Project. J Allergy ClinImmunol. 2008; 122 (6): 1161-1165.
- 21. Yocum M.W., Butterfield J.H., Klein J.S. et al. Epidemiology of anaphylaxis in Olmsted County: A populationbased study. J Allergy ClinImmunol. 1999; 104 (2 Pt 1): 452-456.
- 22. Kopper R.A., Odum N.J., Sen M. et al. Peanut protein allergens: the effect of roasting on solubility and allergenicity. Int Arch Allergy Immunol. 2005; 136 (1): 16-22.
- 23. Du Toit G., Katz Y., Sasieni P. et al. Early consumption of peanuts in infancy is associated with a low prevalence of peanut allergy. J Allergy ClinImmunol. 2008; 122 (5): 984-991.
- 24. Sampson H.A. Update on food allergy. Journal of Allergy and Clinical Immunology. 2004; 113 (5): 805-819.
- 25. Bauer A., EkanayakeMudiyanselage S., WiggerAlberti W. et al. Oral rush desensitization to milk. Allergy. 1999; 54 (8): 894-895.
- **26.** Patriarca G., Buonomo A., Roncallo C. et al. Oral desensitization in cow milk allergy: immunological findings. Int J ImmunopatholPharmacol. 2002; 15 (1): 53-58.
- 27. Patriarca G., Nucera E., Roncallo C. et al. Oral desensitizing treatment in food allergy: clinical and immunological results. Aliment PharmacolTher. 2003; 17 (3): 459-465.
- 28. Buchanan A.D., Green T.D., Jones S.M. et al. Egg oral immunotherapy in non anaphylactic children with egg allergy. J Allergy ClinImmunol. 2007; 119 (1): 199-205.
- 29. Staden U., RolinckWerninghaus C., Brewe F. et al. Specific oral tolerance induction in food allergy in children: efficacy and clinical patterns of reaction. Allergy. 2007; 62 (11): 1261-1269.
- 30. Longo G., Barbi E., Berti I. et al. Specific oral tolerance induction in children with very severe cow's milkinduced reactions. J Allergy ClinImmunol. 2008; 121 (2): 343-347.

ACADEMICIA

ISSN: 2249-7137 Vol. 11, Issue 5, May 2021

- **31.** Skripak J.M., Nash S.D., Rowley H. et al. A randomized, doubleblind, placebo-controlled study of milk oral immunotherapy for cow's milk allergy. J Allergy ClinImmunol. 2008; 122 (6): 1154-1160.
- **32.** Clark A.T., Islam S., King Y. et al. Successful oral tolerance induction in severe peanut allergy. Allergy. 2009; 64 (8): 1218-1220.
- **33.** Jones S.M., Pons L., Roberts J.L. et al. Clinical efficacy and immune regulation with peanut oral immune- therapy. J Allergy ClinImmunol. 2009; 124 (2): 292-300. 300 e291-297.
- **34.** Blumchen K., Ulbricht H., Staden U. et al. Oral peanut immunotherapy in children with peanut anaphylaxis. J Allergy ClinImmunol. 2010; 126 (1): 83-91 e81.
- **35.** Allam J.P., Wurtzen P.A., Reinartz M. et al. Phl p 5 resorption in human oral mucosa leads to dose-depend- ent and time-dependent allergen binding by oral mucosal Langerhans cells, attenuates their maturation, and enhances their migratory and TGF-beta1 and IL-10-producing properties. J Allergy ClinImmunol. 2010; 126 (3): 638-645 e631.
- **36.** Mempel M., Rakoski J., Ring J. et al. Severe anaphylaxis to kiwi fruit: Immunologic changes related to successful sublingual allergen immunotherapy. J Allergy ClinImmunol. 2003; 111 (6): 1406-1409.
- **37.** Kerzl R., Simonowa A., Ring J. et al. Life threatening anaphylaxis to kiwi fruit: protective sublingual allergen immunotherapy effect persists even after discontinuation. J Allergy ClinImmunol. 2007; 119 (2): 507-508.
- **38.** Enrique E., Pineda F., Malek T. et al. Sublingual immunotherapy for hazelnut food allergy: a randomized, doubleblind, placebo controlled study with a standard ized hazelnut extract. J Allergy ClinImmunol. 2005; 116 (5): 1073-1079.
- **39.** Hofmann A.M., Scurlock A.M., Jones S.M. et al. Safety of a peanut oral immunotherapy protocol in children with peanut allergy. J Allergy ClinImmunol. 2009; 124 (2): 286-291.
- **40.** Varshney P., Steele P.H., Vickery B.P. et al. Adverse reactions during peanut oral immunotherapy home dos ing. J Allergy ClinImmunol. 2009; 124 (6): 1351-1352.
- **41.** Staden U., RolinckWerninghaus C., Brewe F. et al. Specific oral tolerance induction in food allergy in children: efficacy and clinical patterns of reaction. Allergy. 2007; 62 (11): 1261-1269.
- **42.** Blumchen K., Ulbricht H., Staden U. et al. Oral peanut immunotherapy in children with peanut anaphylaxis. J Allergy ClinImmunol. 2010; 126 (1): 83-91.
- **43.** Skripak J.M., Matsui E.C., Mudd K. et al. The natural history of IgE mediated cow's milk allergy. J Allergy ClinImmunol. 2007; 120 (5): 1172-1177.
- **44.** Savage J.H., Matsui E.C., Skripak J.M. et al. The natural history of egg allergy. J Allergy ClinImmunol. 2007; 120 (6): 1413-1417.