

# CLINICAL AND PATHOGENETIC ASPECTS OF NUTRITIONAL DISORDERS IN YOUNG CHILDREN

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## Abstract

Nutritional disorders in young children develop on the basis of complex pathogenetic mechanisms arising from physiological immaturity of the organism. The article scientifically analyzes morphofunctional features of the digestive tract, maturation processes of enzymatic systems, neurohumoral regulation mechanisms and the role of intestinal microbiota. Clinical syndromes are pathogenetically substantiated with their age-specific course characteristics demonstrated. Based on research findings, practical directions for diagnostics and prophylaxis are proposed.

**Keywords:** hypotrophy, malabsorption, fermentopathy, dysbacteriosis, mucositis, hypolactasia, exicosis, aminoacidopathy, enterocyte, dysbiocenosis, malnutrition, polyhypovitaminosis, pancreatitis, enteritis, colonocyt

## Introduction

### Literature Review

Various approaches exist in studying the pathogenesis of nutritional disorders. Baranov A.A. and Shcherbakova M.Yu. analyzed the maturation processes of the digestive system, distinguishing stages of postnatal development of enzymatic systems. Their research clearly demonstrates age-dependent changes in lactase and pancreatic enzyme activity, although it relegates the role of microbiota to secondary importance. Zaprudnov A.M. and Grigoriev K.I. emphasized the clinical significance of dysbiotic conditions, presenting intestinal microbiota disruption as the primary etiopathogenic factor. While this perspective holds practical value, it provides less coverage of the complex interactions between enzymatic and immune mechanisms. Uzbek scientists Mirzaeva M.S. and Abdullayev T.R. investigated the characteristics of nutritional disorders in local conditions. Their work demonstrated the influence of dietary habits and socioeconomic factors, though pathogenetic mechanisms were not sufficiently elaborated. Khaitov R.M. evaluated immune system immaturity as an important link in the development of nutritional disorders and identified the relationship between the functional state of intestinal lymphoid structures and clinical manifestations. Korovina N.A. and Zakharova I.N. developed a classification of enzyme deficiencies and substantiated their clinical polymorphism.

### Methodology

The clinical presentation of nutritional disorders manifests differently depending on age, etiopathogenic factors, and process duration. The most widespread clinical manifestation is hypotrophy, characterized by body weight lagging behind age-appropriate indicators. In first- and second-degree hypotrophy, reduction of skin and subcutaneous fat layers, decreased turgor, general weakness, and slowing of psychomotor development are observed. Malabsorption syndrome



develops as a result of disrupted digestion and absorption processes of various food components. Clinically, this syndrome manifests through excessive stool volume compared to consumed product quantity, altered color, and malodorous feces. Undigested food residues, fat droplets, and foam formation are observed in the stool. Enzyme deficiencies, particularly hypolactasia, manifest after milk consumption with abdominal pain, increased gas accumulation, and liquid stools. This condition becomes particularly evident from the third month of life, as the proportion of dairy products in the diet increases during this period. In dysbiotic conditions, stool character changes, constipation and diarrhea alternate, and discomfort appears in the abdominal cavity. Allergic skin reactions and general irritability are frequently observed.

The pathogenesis of nutritional disorders is multifaceted and encompasses several fundamental mechanisms. Morphofunctional immaturity of the digestive tract constitutes the primary pathogenetic factor. Following birth, villous structure of the intestinal mucosa and differentiation of enterocytes and goblet cells continue. Complete finalization of these processes occurs by age two. Enzymatic system maturation processes occur within specific time intervals. Pancreatic amylase, lipase, and proteolytic enzyme activity increases gradually during the postnatal period. Disaccharidases, including lactase, maltase, and sucrase, are synthesized in the intestinal mucosa, and their activity is regulated through mechanisms adapting to nutritional composition. Extremely low or absent lactase activity is termed hypolactasia and is often inherited. Neurohumoral regulation mechanisms also hold significant importance in nutritional disorder development. Motility regulation accomplished through the vagus nerve and sympathetic innervation remains imperfect in early age. Production and receptor effects of gastrin, secretin, cholecystokinin, and other gastrointestinal hormones remain incomplete. Intestinal microbiota formation begins from the first days of life and achieves relatively stable conditions by age three. Normal ratios of bifidobacteria, lactobacilli, and *Escherichia coli* ensure nutrient absorption, accomplish vitamin synthesis, and support local immunity. Disruption of this balance leads to dysbiocenosis development and nutritional disorders. Immune system immaturity also plays an important role. The functional state of intestinal lymphoid structures, particularly Peyer's patches and lamina propria lymphocytes, determines responses to infections and nutritional antigens. Reduced secretory immunoglobulin A levels decrease mucosal protective function. Amino acid and vitamin metabolism disruption develops as a result of malabsorption. Essential amino acid deficiency reduces protein biosynthesis and slows growth processes. Fat-soluble vitamin deficiencies, particularly vitamins A, D, E, and K, manifest through various clinical signs.

### Results

Analysis of nutritional disorder pathogenesis demonstrates that this process is multifactorial, encompassing several pathological links. Morphofunctional immaturity, changes during enzymatic system maturation stages, neurohumoral regulation imperfections, and microbiota imbalance collectively determine clinical manifestations. Diagnostic approaches must include clinical manifestations, anthropometric indicators, laboratory assessment of enzyme activity, and coprological analyses. Molecular genetic examinations prove valuable in identifying inherited enzyme deficiencies. Therapeutic strategy must be pathogenetically grounded and incorporate enzymatic preparations, probiotics, nutritional correction, and vitamin therapy. Preventive measures



importantly include rational maternal nutrition during pregnancy, proper introduction of complementary foods during breastfeeding, and regular monitoring of physical development.

### Discussion

While the obtained results demonstrate general concordance with data from other researchers, differences exist in certain aspects. Baranov A.A. research emphasized enzymatic immaturity playing the primary role, whereas our analysis considers the interconnection of microbiota and immune mechanisms more worthy of attention. The question regarding the significance of dysbiocenosis remains debatable. Some authors consider it an independent pathological process, while others evaluate it as a secondary phenomenon. In our opinion, assessing microbiota status not in isolation but in conjunction with other pathogenetic mechanisms represents the correct approach. Diagnostic criteria indicate the necessity for standardization, as various authors apply different approaches. Unified protocols must be developed for determining the degree of enzymatic insufficiency.

Nutritional disorders in early childhood develop based on complex pathogenetic mechanisms and require comprehensive clinical assessment. Morphofunctional immaturity, incomplete enzymatic system maturation, neurohumoral regulation imperfections, and microbiota imbalance constitute interconnected pathological links. Diagnosis requires a comprehensive approach, while therapy demands pathogenetic substantiation. Preventive measures must be implemented from the first days of life.

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