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CHANGES IN THE ACTIVITY OF SMALL INTESTINAL DISACCHARIDASES IN DIABETES MELLITUS

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ABSTRACT:

Diabetes mellitus (DM) is a chronic metabolic disease associated with insufficient insulin secretion or impaired insulin action. DM not only disrupts the metabolism of carbohydrates, fats, and proteins but also seriously affects the function of the digestive system. The aim of this article is to study the mechanisms and clinical significance of decreased activity of key disaccharidase enzymes (lactase, sucrase, maltase) located in the small intestine of patients with DM. Research results indicate that hyperglycemia, oxidative stress, and tissue insulin resistance lead to morphological and functional impairments of the small intestinal epithelium, which causes a decrease in enzyme activity and, consequently, impaired digestion of disaccharides (disaccharidase deficiency). This condition leads to the exacerbation of gastrointestinal symptoms such as abdominal pain, diarrhea, and flatulence in patients.

Keywords: Diabetes mellitus, small intestine, disaccharidases, lactase, sucrase, maltase, disaccharidase deficiency, hyperglycemia.

INTRODUCTION

The global prevalence of diabetes mellitus is increasing annually. While the complications of the disease, including nephropathy, neuropathy, and retinopathy, are well studied, changes in the gastrointestinal system (diabetic enteropathy) are relatively less explored. The small intestine is the primary site for the digestion and absorption of food, and the enzymes located in its brush border (disaccharidases and peptidases) play a crucial role in the breakdown of carbohydrates and proteins. Recent research has been accumulating data on the decreased activity of these enzymes in DM and its negative impact on the patient's nutritional status and quality of life.

1. Biological Significance of Disaccharidases

Disaccharidases (lactase, sucrase-isomaltase complex, glucoamylase/maltase), located in the microvilli of small intestinal enterocytes, break down disaccharides (sucrose, lactose, maltose) into monosaccharides. For example, lactase breaks down lactose into glucose and galactose, while sucrase breaks down sucrose into glucose and fructose. Only monosaccharides can be absorbed. Therefore, a decrease in disaccharidase activity sharply reduces the efficiency of nutrient utilization from food. Hormonal imbalance, microangiopathy, oxidative stress, and impaired trophism of the mucosal layer observed in diabetes lead to a weakening of enzyme activity.

Research shows that in diabetes:

- Even if pancreatic enzymes are synthesized sufficiently, their activation in the intestine is inadequate (due to decreased enterokinase secretion);
- Disaccharidase expression in the mucosal layer is damaged by glycation due to chronically high glucose levels;

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- Insulin also acts as a trophic hormone that enhances protein synthesis, renewal, and enzyme transcription in the intestinal epithelium; its deficiency exacerbates enterocyte dysfunction;
- Oxidative stress causes damage to enterocyte membranes and enzyme active sites;
- Dysbiosis of the gut microbiota alters the metabolism of enzyme substrates.

2. Mechanisms of Decreased Enzyme Activity in Diabetes Mellitus

Direct Effect of Hyperglycemia: High blood sugar levels activate glycation processes in cells. In this process, sugar molecules in the blood react with enzyme and other protein molecules, disrupting their structure and function. The decreased activity of GLUT-2 transporters results in a reduced energy source necessary for enterocytes.

Oxidative Stress and Inflammation: DM increases the production of free radicals in the body and weakens the antioxidant defense system. Oxidative stress leads to apoptosis (programmed cell death) of enterocytes and disrupts the structure of microvilli.

Autonomic Neuropathy: Diabetic neuropathy often disrupts the innervation of the small intestine. This leads to slowed intestinal motility (hypermotility or hypomotility), increased bacterial growth (SIBO), and, consequently, damage to the mucosal layer.

Microangiopathy of Microvessels: Damage to the capillaries in the small intestinal wall impairs the nutrition and oxygen supply to the tissues, which reduces the functional activity of enterocytes.

* Decreased breakdown of disaccharides → accumulation of substrate in the intestinal lumen → malabsorption, osmotic diarrhea;
* Impaired protein digestion → hypotrophy, flatulence;
* Irregular glucose absorption → increased glycemic variability.

The decrease in small intestinal enzymatic activity is a secondary consequence of diabetes, with **insulin deficiency** and **microcirculatory disorders** being the most important factors exacerbating this process. To alleviate this condition in clinical practice, the following are appropriate:

- Strict control of glucose,
- Optimization of insulin therapy,
- Use of antioxidant and pro-trophic approaches,
- Administration of enzyme preparations to facilitate digestion.

Conclusion

Diabetes mellitus is a significant factor that reduces the activity of disaccharidase enzymes in the small intestinal brush border. This impairment occurs through a number of pathophysiological mechanisms such as hyperglycemia, oxidative stress, neuropathy, and angiopathy. The resulting disaccharidase deficiency negatively affects the patient's quality of life and can further complicate blood sugar control. Therefore, in the treatment of patients with DM, it is necessary not only to control glycemia but also to early identify and correct gastrointestinal complications, including enzyme deficiency (for example, with enzyme preparations and diet).

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