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INTESTINAL MICROBIOTA AND METABOLISM: THEIR BIOCHEMICAL ROLE AS A "NEW ENDOCRINE ORGAN" IN THE HUMAN BODY

Suyunov Farrux Sayfullo o'gli

5th-year student of the Pediatric Faculty of Samarkand State Medical University

Pardayeva Azizabonu Ulug'bek qizi

3-rd year student of the Faculty of General Medicine of Samarkand State Medical University

Sulaymonova Zebiniso Ilyos qizi

5th-year student of the Pediatric Faculty of Samarkand State Medical University

Adxamov Asror Adxam o'g'li

5th-year student of the Faculty of Medicine of Samarkand State Medical University

Abstract

The intestinal microbiota is recognized as an important regulator of metabolism in the human body. The metabolites it produces - short-chain fatty acids (SCFA), signaling molecules that activate incretin secretion, and neuroactive substances - regulate energy metabolism, insulin sensitivity, and central nervous system function. Disruption of the microbiota composition leads to a weakening of the intestinal barrier function, the development of low-grade chronic inflammation, and insulin resistance. This article analyzes the main molecular mechanisms of microbiota's influence on metabolism.

Keywords

intestinal microbiota, SCFA, GLP-1, insulin resistance, obesity, brain-intestinal axis, metabolic syndrome.

Introduction

In recent years, metabolic diseases - obesity and type 2 diabetes - have become a global epidemic. According to the classical model, these diseases were considered as a disruption of the energy balance associated with excess calories. However, a sharp difference in the metabolic response in people with the same diet indicated the presence of a new pathogenetic factor.

The development of molecular biology and genomics has revealed the active participation of intestinal microorganisms in the regulation of metabolism. Metabolites produced by the intestinal microbiota control hormonal signal transmission, immune response, and central nervous system function. Therefore, the microbiota is now considered an independent metabolic regulator and a "new endocrine organ."

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Intestinal microbiota and its metabolic function

More than 100 trillion microorganisms live in the human gut. Their genetic potential is much greater than that of the human genome, and they expand metabolic reactions:

• breaks down indigestible polysaccharides.

• synthesizes vitamins

• produces signaling molecules

• manages energy balance

The microbiota works in symbiosis with the host organism: the host provides food, and the bacteria provide metabolic control.

Short-chain fatty acids (SCFA)

When bacteria ferment fibres, the following metabolites are produced:

• Acetate

• Propionate

• Butyrate

Biochemical effect

SCFA transmits signals through G-protein receptors and reprograms metabolism:

1. Reduces hepatic gluconeogenesis

2. Increases glucose utilization in muscles

3. Increases fat oxidation

4. Reduces inflammation

Special role of butyrate

Butyrate is the main source of energy for colonocytes, strengthening the intestinal barrier. When it decreases, bacterial endotoxins enter the bloodstream, and metabolic inflammation develops.

GLP-1 secretion and mechanism of incretin

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Enteroendocrine L-cells in the intestine produce the hormone GLP-1.

SCFA activates these cells.

Result

• increased insulin secretion.

• glucagon decreases.

• stomach relaxation slows down.

• a feeling of satiety appears.

Thus, the microbiota regulates sugar metabolism before the pancreas.

Development of insulin resistance

In the case of dysbiosis, gram-negative bacteria multiply and release lipopolysaccharide (LPS).

LPS → immune response → inflammatory cytokines → insulin signal blocked

At the molecular level:

• IRS-1 phosphorylation is impaired

• GLUT-4 translocation decreases

As a result, the cell does not receive glucose - insulin resistance develops.

Obesity association

In obesity, the composition of the microbiota changes and energy release increases.

Mechanisms:

• calorie release from food increases

• lipogenesis increases.

• accumulates fat

• the incretin signal changes.

Therefore, obesity is not just an overeating, but a metabolic signaling disease.

Cerebrointestinal axis

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Intestinal microbiota communicates with the central nervous system in three ways:

1. Nerve duct - vagus nerve

2. Endocrine pathway - GLP-1, PYY, serotonin

3. Immune pathway - cytokines

Bacteria produce neurotransmitter precursors, which:

• appetite

• stress

• mood

• controls the choice of food.

Discussion: Explaining metabolic disorders solely as impaired glucose metabolism is insufficient. The microbiota is a high-level regulator of the body's energy metabolism. Dysbiosis leads to the development of metabolic syndrome through disruption of the intestinal barrier and chronic inflammation.

Therefore, in modern therapy, microbiota modulation - prebiotics, probiotics, and dietary fiber - is becoming a promising area for the treatment of metabolic diseases.

CONCLUSION

The intestinal microbiota functions as an independent endocrine system that regulates metabolism. Through SCFA production, GLP-1 secretion, and the brain-intestinal axis, it controls energy balance, insulin sensitivity, and eating behavior. Disruption of the microbiota is an important pathogenetic mechanism for the development of insulin resistance and obesity. In the medicine of the future, it is expected that the treatment of metabolic diseases will be aimed at restoring the microbiota.