International Journal of PharmATA

2022; 2(2): 68-71

Review

The Role of Gut Microbiota on Appetite Hormones

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ABSTRACT:

The fact that obesity has reached alarm level worldwide shows that this problem has practical and manageable limitations, both prevention and treatment options. It is known that obesity is effective in the emergence of many diseases such as asthma, depression and cancer, especially cardiometabolic diseases. Obesity, which shows a complex development, is mainly due to the chronically positive energy homeostasis. Intestinal microbiota plays a key role in maintaining this homeostasis. Microbiota is called all the microorganisms in the human body, and it acts as an organ by contributing directly or indirectly to many metabolic, structural and protective physiological functions of the host. Many of these functions are mediated by short-chain fatty acids (SCFA) produced from carbohydrates that are resistant to digestion by the microbiota. SCFA (butyrate, propionate, acetate) activate some hormone and signaling pathways and have a significant effect on appetite and body weight regulation. In this review, the effects of SCFAs on some gastrointestinal appetite hormones will be explained and predictions will be shared about their role in metabolism and their potential to be used as new molecular targets.

Obesity is one of the serious public health problems due to its increasing prevalence globally and its contribution to common metabolic disorders. The main goal of the methods used in the treatment of obesity is to reduce food intake by providing appetite regulation. SCFAs produced by the microbiota regulate appetite by decreasing food intake and increasing energy expenditure, anorexigenic hormones PYY and GLP-1.

Keywords *:* Appetite hormones, gut microbiota, short-chain fatty acids.

Received	Accepted	Published
30.03.2022	02.04.2022	06.04.2022

To cite this article:

Alyar G. The Role of Gut Microbiota on Appetite Hormones. International Journal of PharmATA. 2022; 2(2); 68-71.

1. INTRODUCTION

Obesity is defined as the excessive accumulation of fat in the body as a result of the deterioration of the energy balance [1]. Recently, the role of gut microbiota has come to the fore in the pathogenesis of obesity, which shows a complex development multifactorially. Microbiota is called all the microorganisms in the human body, and it acts as an organ by contributing directly or indirectly to many metabolic, structural and protective physiological functions of the host [2]. Many of these functions are mediated by short-chain fatty acids (SCFA) produced from carbohydrates that are resistant to digestion by the microbiota. SCFAs (butyrate, propionate, acetate) have a significant effect on maintaining appetite and body weight regulation by activating some hormone and signaling pathways [3]. SCFAs are generally produced by digestion-resistant carbohydrates and serve as an important substrate for gluconeogenesis. Acetate, propionate and butyrate from SCFAs produced in different amounts are important in

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ensuring energy regulation by acting on a number of hormones [4]. Acetate is transported to the liver via the portal vein after intestinal absorption and distributed throughout the body, where it serves as a substrate for cholesterol synthesis. Propionate, on the other hand, is used as the primary substrate in gluconeogenesis by reaching the liver through the portal circulation after intestinal absorption [3,5]. The physiological effects of other SCFA butyrate are quite remarkable. As a result of in vitro studies, it has been determined that 70% of the energy needs of intestinal epithelial cells and colonocytes are met by butyrate [6]. In addition, butyrate plays a role as a regulator in epithelial cell growth and differentiation. At the same time, ~10% of the host's daily energy requirement is provided by butyrate [7]. In particular, butyrate has positive effects such as the differentiation of beta cells, proliferation and prevention of beta cell apoptosis, similar to histone deacetylase inhibitör [8]. In addition to all these, butyrate glycemic control; It increases insulin transcription and translation as a result of induction of signaling pathways directly or indirectly, and has a positive effect by inhibiting gluconeogenesis and glycogenolysis in the liver (via indirect glucose production) [9].

2. DISCUSSION

Microbiota is called all the microorganisms in the human body, and it acts as an organ by contributing directly or indirectly to many metabolic, structural and protective physiological functions of the host [10]. Many of these functions are mediated by short-chain fatty acids (SCFA) produced from carbohydrates that are resistant to digestion by the microbiota [11]. The fact that the total amount of SCFA (butyrate, propionate, acetate) is higher in obese people than in lean individuals and the decrease in fecal SCFAs in treated obese individuals indicates that SCFAs may contribute to obesity [12]. To determine the effects of SCFAs on appetite hormones, we focused on the expression and function of the receptors of GPR41 and GPR43 in subtypes of enteroendocrine cells such as L and K cells [13]. The anorexigenic hormones PYY and GLP-1 activated through GPR41 and GPR43 reduce food intake and increase energy expenditure and provide appetite regulation.In addition, hormones increase satiety by delaying gastric emptying [12]. In vitro and in vivo studies have shown that SCFAs activate gastrointestinal satiety hormones PYY and GLP-1 [7,8,9,10,11]. Indigestible carbohydrates found in dietary fibers promoted the growth of SCFA-producing strains, resulting in metabolic improvement in individuals with obesity [14]. However, the amount of SCFA increased with the change in the microbial composition of the mice administered probiotics. This increase induces the release of the GLP-1 hormone, resulting in a decrease in food intake and an increase in glucose tolerance [15]. In the studies, intestinal microbial dysbiosis and the development of diet-related obesity were prevented by the supplementation of SCFA [6, 13]. In addition, free SCFAs cross the blood-brain barrier as monocarboxylate transporters, allowing the gut to act as signaling molecules to transmit the state to the brain [4].

Conflict of Interest

Author has no personal financial or non-financial interests.

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