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# The Pivotal Role of Microbiota in Obesity

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**Abbreviations**

BMI: Body Mass Index, FIAF: Fasting-Induced Adipocyte Factor, LPS: Lipopolysaccharides

Obesity is a chronic disease defined as body mass index (BMI) of ≥30 kg/m$^2$. Both obesity and overweight (BMI of 25 to 29.9 kg/m$^2$) represent a major public health issue worldwide in children, adolescents and adults leading to increased morbidity and mortality. In general, obesity is a complex disease that results from the interplay between several determinants. From time to time, a role for host genetics, environment, biological factors, pregnancy and lactation history in mothers, social and cultural influence, and imbalance between energy intake and expenditure has been advocated [1-4]. Dietary factors modulate the composition of the gut microbiota [5].

Recently, an increased number of studies have evaluated the role of intestinal microbiota in the development of the epidemic obesity. Intestinal microbiota play a pivotal role in regulating endogenous energy and metabolic pathways [6]. Alterations in gut microbiota have been observed in obese animals and humans according to phylum-level, bacterial diversity and representation of bacterial genes and metabolic pathways [7,8]. In this respect, an increase in Firmicutes associated with reduction in Bacteroidetes is observed in obesity [9,10]. The analysis of the faecal microbiota of obese and lean twins have shown that both subjects displayed the core gut microbiome, but obese individuals were characterized by reduced microbial diversity and abnormal microbiota metabolic pathways [11].

Intestinal microbiota influences energy harvest and storage [12], since microbiota can regulate nutrient absorption and host genes [13]. Microbiota might modulate energy balance through several pathways: an increased intestinal absorption of monosaccharides and the production of short-chain fatty acids through the fermentation of indigestible polysaccharides, an increased hepatic lipogenesis via carbohydrate and sterol response-element binding proteins, a suppression of Fasting-Induced Adipocyte Factor (FIAF) that in normal conditions inhibits lipoprotein lipase. Additional mechanisms include suppression of adenosine monophosphate-activated protein kinase, interplay between products of polysaccharide fermentation and G-protein-coupled receptor 4, increased intestinal permeability for bacterial Lipopolysaccharides (LPS) in response to the consumption of high-fat diets leading to an elevated systemic LPS level and low-grade inflammation [14].

To date, the accumulating evidences suggest that the gut microbiota contribute to adiposity by modulating the metabolic network involved in bioenergetics. Thus, manipulation of intestinal microbiota represents a novel and fascinating approach to shift a dysmetabolic status into a healthy condition. Moreover, by modifying host energy balance and treating obesity is a chance to prevent other metabolic diseases.

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