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EDITORIAL

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Gut microbiota: Enemy inside the obese

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Obesity-associated disorders increasing at an alarming rate. The most prominent causes are the sedentary lifestyle and increased consumption of food. Environmental, genetic factors and gut microbiota plays a complex role in the development of metabolic disorders. [1, 2] The human body contains a large number of microorganisms, which invade the intestinal tract during parturition (maternal vaginal microbes), by the maternal skin, and during breastfeeding. [3] Gut microbiota can be classified into four major phyla - Firmicutes, Bacteroidetes, Actinobacteria, and Proteobacteria. The large intestine is the main host for gut microbiota; over 10^{11} bacteria are present in each gram of intestinal content. Gut microbiota plays a vital physiological role, which includes digestion, synthesis of new biomolecules and metabolism amongst others. Gut microbiota is linked to increase in dietary energy production, regulation of fatty acid tissue composition and low-grade inflammation, triggering obesity. [4]

Researches in the recent years revealed the close association of gut microbiota and obesity. The difference between gut microbiota of obese and lean mice is well established. A shift of bacteria from Bacteroidetes phyla towards Firmicutes phyla is closely related with augmented energy absorption from diet and initiates low-grade inflammation. [5] Colonic fermentation of digestive products like dietary fiber, proteins by gut microbiota produces short-chain fatty acids (SFCAs). Cecal and fecal level of this biomolecule is found more in genetically obese ob/ob mice and obese individuals connected with reduced colonic absorption. [6] SCFA act as signaling molecules in liver and muscle activates AMP-activated protein kinase (AMPK) cholesterol, lipid, and glucose metabolism by a variety of receptors namely peroxisome proliferator-activated receptor-gamma coactivator 1 alpha (PGC-1α), Peroxisome proliferator-activated receptor gamma (PPARγ), and Liver X receptors (LXR). [7] This activates low-grade inflammatory response, which leads to obesity. Higher level of lipopolysaccharides (LPS) also increases fat intake. Toll-like receptors (TLRs) and nuclear factor kappa (NF-kB) are the key factors for the genesis of pro-inflammatory cytokines by a cascade pathway. [8] The production of LPS activates this pathway. Interestingly LPS is found in the outer membrane of Gram-negative bacteria produced in the gut. Gut microbiota also regulates bile acids and cholesterol metabolism by up-regulating several transcription factors connected with

nutritional-induced inflammation, absorption of lipid and lipogenesis. [9]

Evidence from emerging research on energy homeostasis and inflammation highlights gut microbiota as a crucial factor in obesity. Prebiotic and probiotic bacteria can be utilized as a treatment option for metabolic diseases in near future.

Abbreviations

AMP-activated protein kinase (AMPK), lipopolysaccharides (LPS), liver X receptors (LXR), nuclear factor kappa (NF-kB), peroxisome proliferator-activated receptor gamma (PPARγ), peroxisome proliferator-activated receptor-gamma coactivator 1 alpha (PGC-1α), short-chain fatty acids (SFCAs), toll-like receptors (TLRs)

Competing interests

None declared.

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