

Altered Intestinal Microbiota by Diet and their Roles on Host Metabolism

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Diet can supply various nutrients for maintaining metabolism and basic activities of organism, such as cell proliferation, apoptosis, and reproduction. The component of dietary affect host immunity and health, and nutrient deficiency in short-term or long-term, often results in corresponding disease. It is highlighted to study the interactions of diet, intestinal microbiota and host. Here, the roles of intestinal microbe should be emphasized, because it interplays like a moderator between diet and host [1].

The intestinal lumen resides with enormous number of symbiosis bacteria, and they are regarded as bio-barrier function against foreign substances. Indeed, the microbiota are involved in nutrient metabolism [2], host response pathway, and surveillance of immune system [3].

Components of diet shape the microbiota communities and function in gastrointestinal tract. The ingredients and nutrient levels in diet both possible affect the diversity of gut microbiota. For instance, low dietary fiber leads to the loss of species reliant of bacteria and reduction in bacterial metabolites of fermentation of end-products [4]. High level of dietary protein can reduce the *Roseburia/E. rectale* group, which is associated with butyrate production [5]. Of course, the nutrient in diet including carbohydrate, protein, fat, vitamins, minerals, and additive might be metabolized through specific microbes. In large intestine, the abundance of bacteria is involved in amino acids metabolism mainly including *Bacteroides*, *Propionibacterium*, *Streptococcus* and *Clostridium*, whereas the dietary fiber is degraded by *Firmicutes* and *Bacteroides* [6]. These findings may help to understand the diversity of intestinal bacteria in different gut sections.

How altered intestinal microbiota through diet modulate host health and disease have been widely investigated. The changes of gut microbiota respond to host by influencing nutrient metabolism and improving innate immunity. The intestinal microbiota communities are closely associated with nutrient digestion and absorption of diet, storage of calories, as well as production of bacterial metabolites. Short-chain fatty acids (SCFA) are one of major bacterial metabolites, which play pivotal roles in supplying energy for epithelium and maintaining mucosal integrity [7]. SCFA metabolism on host is related to G-protein coupled receptor 41 and 109A, which might be regulated by gut-microbiota-brain-endocrine axis [8]. Some of bacterial metabolites with high concentration, such as polyamines and hydrogen sulfide, have detrimental effects on epithelial cell frequently resulting in diseases related to dys-homeostasis of microorganism in gut.

Mucus layer serves as chemical barrier function against pathogenic microorganisms in lumen. The major component of this viscoelastic gel is mucin, which secreted by goblet cells with characteristics of heavily glycosylated proteins rich in serine, threonine and proline. The intestinal commensal bacteria residue in the outer mucus layer of intestine lumen and closely interact with mucin. It has been well documented that the cross-talk between gut microbiota and mucin play an important role in gut-associated immune system [9]. The diet, such as fiber, amino acids, mineral and plant extraction can alter the mucin secretion and their physiological functions. This may due to modulation of intestinal microbiota by diet. The microbiota is able to use amino acids original from diet to synthesize special protein, e.g. threonine, for mucin secretion of goblet cells in host. Meanwhile, the intestinal bacteria can utilize the mucins, particularly the oligosaccharides as nutrients for bacterial growth.

Recently, many studies have focused on the nutrient metabolomics, intestinal microbiome, proteomics and computation [10]. The integrated-data analysis via these advance methods may contribute to reveal the functions of gut microbiota, and underlying mechanism of nutrient modulation on intestinal microbiota and host reaction.

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