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Interactions of Gastrointestinal Microbiota and Mammalians Health

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Abstract

The gut microbiota is linked to intestinal epithelial cell metabolism and influences health of the host. Gut microbial disorders influence host health, as exemplified by the associations with metabolic syndrome, diarrhea obesity-related disease and cancer. Intake of dietary fiber or plant-based foods can alter gut microbiota and then modulate these disorders. This review summary the relationship between the gut microbiota and healthy, which could assist the development of effective manipulations in enhancing mammality health and productivity.

Keywords: Gastrointestinal tract; Microbiota; Host-microbiota interactions; Meta-omics; Health

Abbreviations: ESCC: Esophageal squamous cell carcinoma; LCA: Lithocholic acid; UDCA: Ursodeoxycholic acid; IGN: Gluconeogenesis; PD: Parkinson's disease; IBS: Irritable bowel syndrome; NAA: N-acetylaspartate

Introduction

Intestinal bacteria have been referred to as the "second brain" in mammalians because they are closely related to brain health. In recent years, many scientists have found that the gut microbiota can affect the host brain structure through a special intestinal-brain axis and regulate the occurrence of many brain-related diseases such as Parkinson's disease (PD), irritable bowel syndrome (IBS), and autism [1-3]. Many metabolites produced by the gut microbiota are absorbed into the blood, and some of them enter the brain by penetrating the blood-brain barrier and affect the brain function, and some appear to regulate immune responses [4-5]. Interestingly, gut microbiome might affect autism [2]. A recent study indicated that gut bacteria "communicate" with the brain metabolites via serum cortisol, as demonstrated by the link between fecal Ruminococcus and brain N-acetylaspartate (NA) [6]. These observations suggest a potential mechanism explaining some of the characteristics of autism. Enteric neurons trigger intestinal movements and transmit gut signals to the nervous system Enterochromaffin cells, accounting for <1% of cells secreted by the gut epithelium, are responsible for producing 90% of the serotonin [4]. A recent study using an intestinal organoid model showed that epithelial cells were electrically excitable and could lead to voltage-gated Ca2+ channel-dependent serotonin release and modulate sensory nerves via serotonin receptors and synaptic connections [7]. This research illuminated a molecular mechanism involving "dialogue" between the gut and the brain. Together, the above studies demonstrate the close relationship between gut microbiota and the development of host diseases.

The presence of some bacteria in the GI tract is directly related to specific diseases. For example, the occurrence of P. gingivalis infection in the epithelium of the esophagus was associated with ESCC [8]. P. gingivalis is more abundant in specimens of esophageal cancer and dysplasia of the esophagus than in cardia or stomach cancers, possibly because this microorganism cannot survive in the highly acidic condition, such as in stomach [9]. These findings suggest that P. gingivalis infection could be a biomarker for this disease and indicate that eradication of this common oral pathogen could potentially contribute to reduction in esophageal cancer [8-10]. The research should be directed to these bacteria-associated cancers, although the emergence of cancer cells is complex involving inheritance, environment, diet, individual immunity, and other factors. The risk of gastrointestinal cancer may be lowered through formulating healthy diets that can modulate the GI microbiota.

Obesity and metabolic syndrome could be the results of disordered interactions between gut microbiota, host genetics, environmental conditions, and diet. In permissive genetic backgrounds, environmental reprogramming of the microbiota could ameliorate obesity and metabolic syndrome [11-12]. Oral administration of Parabacteroides distasonis can alleviate obesity and metabolic dysfunctions in an obese mouse model. Lithocholic acid (LCA), ursodeoxycholic acid (UDCA), and succinate were significantly elevated in the gut after treatment with live P. distasonis, and LCA and UDCA are known to reduce hyperlipidemia by activating the FXR pathway and repairing gut

barrier integrity, whereas succinate can decrease hyperglycemia in obese mice via the activation of intestinal gluconeogenesis (IGN) [13]. Succinate produced by P. distasonis is important for modulation of the host metabolism because succinate binds to fructose-1,6-bisphosphatase, the rate-limiting enzyme in IGN.

Some microorganisms benefit host health when consumed lived and in adequate quantities (i.e., probiotics). This method has been used in many aspects, and species of Lactobacillus, Bifidobacterium, Streptococcus, Enterococcus, Propionibacteria, and Saccharomyces boulardii are among the commonly used probiotics. Similarly, microbiota transplantation has also been used to improve intestinal health [13-14].

Conclusion

Various microbial metabolites may affect host metabolism and sometimes stimulate abnormal metabolism. Understanding the characteristics of the interactions between gut microbial profile and their specific metabolites and particular host disease could potentially help to diagnose, prevent, and treat host diseases in a specifically targeted fashion. The research on gut microbiota mostly focuses on health, whereas the research on the animal gut microbiota aims to increase productivity as well as to improve health. All of research efforts share a common goal – understanding of the host-microbiota interaction to achieve knowledge-based manipulation of gut microbiota. The bacteria associated with special diseases should be the focus of this research.

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Competing Interests

The authors declare that they have no competing interests

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