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Fecal Microbiome and Bowel Disease

Chien Ming*

Research Institute in Healthcare, Micro Research Centre, UK

Introduction

IBD is a chronic relapsing nonspecific inflammatory condition of the gastrointestinal (GI) tract that includes ulcerative colitis (UC) and Crohn's disease (CD). IBD has an unclear cause. Disturbance of the intestinal microbiota and its metabolites, the host's genetic predisposition, and the host's innate and acquired immunity are all contributors in the pathogenesis of IBD. The normal patterns of the human intestinal commensal microbiota have been profiled in several studies based on metagenomics. 3.3 million bacterial genes, up to 10 bacterial phyla, and over 1000 bacterial species, for example. In the human intestine, commensal fungi and viruses have also been discovered. With the advancement of high-throughput DNA sequencing and bioinformatics analysis capabilities, metagenomic researches have become more common. As a result, much research have focused on the composition and function of gut microbiota in IBD patients, but results on the defined modification of intestinal microbiota, microbial relationships, and host-microbe interactions are still lacking. Furthermore, traditional medical treatments for IBD include aminosalicylates, steroids, immunosuppressive drugs, and biological therapies, all of which have numerous side effects, and there is no cure. In recent years, alternative IBD therapy techniques aiming at repairing the damaged gut microbiota have sparked a lot of attention. The efficacy of probiotics in IBD has been studied in numerous clinical trials. Although various probiotic products can elicit and maintain remission in UC, their positive effect is small.

Description

The alteration of intestinal microbiota associated with IBD has been better documented thanks to the introduction of culture-independent approaches like metagenomic analysis. In IBD remission and relapse, this involves the participation of the feces/colonic mucosa-associated microbiota, inflammatory lesions-/normal mucosa-associated microbiota, and even the intestinal microbiota. Fungal communities are also key components of the microbiota of the human GI tract, with most of them co-evolving in a symbiotic relationship with the host. In a mouse model of dextran-sulfate-sodium-induced colitis, a study recently linked the intestinal fungal microbiota with the host immune system via Dectin-1, confirming the fungal etiology in IBD. Overall, IBD reduces the diversity and abundance of intestinal bacterial microbiota, as well as the bacterial microbiota metabolites. However, because to inter-individual heterogeneity, distinct IBD features or subtypes, and diverse data processing methodologies, a particular IBD microbiome has yet to be discovered. Furthermore, it is still unclear whether microbial pathogenesis is the primary cause of IBD or a secondary cause [1-2]. Furthermore, more research based on fungal high-throughput DNA sequencing is needed to determine if the change in the fungal community structure is a result of the imbalance in the intestinal bacterial community or a result of IBD pathogenic mechanisms that are independent. *Mycobacterium avium* subspecies paratuberculosis, which has been connected to the etiology of CD but without solid proof, can invade the ileal mucosa of CD patients. In addition, CD patients' ileums were shown to contain *Escherichia coli* (*E. coli*) strain LF82. Several studies have found that CD patients had a greater prevalence of adherent-invasive *E. coli* (AIEC) in their ileal lesions, indicating that AIEC has a distinct relationship with CD. Furthermore, aberrant metabolites produced by the gut bacterial microbiota may have a role in the development of IBD. Butyric acid, for example, which is the main energy source of intestinal epithelial cells, can block proinflammatory cytokine signaling.

In an animal model of colitis, butyrate-producing bacteria and their culture supernatants can reduce intestinal inflammation and necrosis. Fungal communities are also key components of the microbiota of the human GI tract, with most of them co-evolving in a symbiotic relationship with the host. When compared to non-IBD controls, there were substantial alterations in fungal communities related to IBD. All IBD patients had fungal sequences in their colonic mucosa, and the variety of the intestinal microbiome grew significantly, although the proportion of microbiome in the total intestinal microbiota was low. Furthermore, it is unknown if the gut microbiota interacts with the mucosal immune system or influences intestinal diseases. In a mouse model of dextran-sulfate-sodium-induced colitis, a study recently linked the intestinal fungal microbiota with the host immune system via Dectin-1, confirming the fungal etiology in IBD [3-5].

Conclusion

There is a growing interest in elucidating the role of the human gut microbiota to determine the therapeutic potential of manipulating it. The introduction of a solution of fecal matter from a donor into the intestinal tract of a recipient to directly affect the recipient's gut microbial composition and provide a health benefit is known as fecal microbiota transplantation (FMT). Recurrent *Clostridium difficile* infection has been successfully treated with FMT. It may also have therapeutic potential for other illnesses such inflammatory bowel disease, obesity, metabolic syndrome, and functional gastrointestinal disorders, according to early evidence.

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***Address for Correspondence:** Chien Ming, Research Institute in Healthcare, Micro Research Centre, UK, E-mail: ming.c01@mic.edu*

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