

Interaction between food, intestinal microbiota and functional GI disorder

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Functional gastrointestinal disorders (FGIDs) are characterized by chronic and recurrent abdominal/visceral symptoms in the absence of identifiable structural or organic lesions. FGID is very common with an estimated prevalence of 10-20% in general population. Though FGID does not lead to serious diseases such as cancer, it does bring a significant burden to our society regarding loss of work productivity, large consumption of medical resources, and impairment of quality of life.

Traditionally, food has been thought to be associated with symptom onset or exacerbation in a significant proportion of FGID patients. Despite of this belief, the role of food in the pathogenesis of the FGIDs has remained poorly understood. Recently, a rapid expansion is noted in our understanding of the role of food in GI function and sensation and how food relates to GI symptoms in FGID patients. Taking carbohydrate diet as an example, a concept has been developed to regard as FODMAPs (fermentable oligosaccharides, disaccharides, monosaccharides and polyols) and to evaluate a dietary approach that restricts FODMAPs. FODMAPs are poorly absorbed in the small bowel and arrive into the colon, populated with microbiota, which will lead to increased osmotic activity followed by increased water retention within the lumen of the small and large bowel. Furthermore, FODMAPs are substrates for colonic bacterial fermentation, which will eventually result in the rapid production of gas and subsequent luminal distension. In patients with irritable bowel syndrome, there is accumulating evidences showing that FODMAPs trigger gastrointestinal symptoms in patients with functional bowel disorders, and that a diet low in FODMAPs offers considerable symptom relief in the majority of patients who use it. Preliminary animal study also demonstrated that diets high in FODMAP caused dysbiosis in the gut accompanied by mucosal inflammation, impaired permeability and visceral hypersensitivity. While low FODMAP reduced intestinal inflammation and improved gut barrier functions, which were associated with normalization of gut sensitivity in rats.

Food restriction/manipulation may provide symptoms improvement in the patients with FGID through the modulation in intestinal microbiota and the associated changes in GI functions. Further research studies are warranted to expand our knowledge of applications and implications of its use.