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New perspectives on probiotics in health and disease

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Abstract

The gut microbiota continues to fascinate scientists in many realms when it is considered that humans contain 90% bacteria. Correlations between changes in composition and activity of the gut microbiota and common disorders such as cancer, hypertension, hypercholesterolemia, inflammatory bowel diseases, obesity, oral health, *etc.* have been proposed. What is the real role of probiotics, prebiotics and synbiotics in influencing a healthy microbiota? Both *in vitro* evidences and *in vivo* clinical data have supported some of these new health claims, while recent molecular advancement has provided strong indications to support and justify the hypotheses. However, probiotics validity and health claims have continuously been rejected on the basis of "biomarker deficiency". To battle the increase in health care costs, a preventive approach to medicine with the development of probiotics and prebiotics or symbiotic products is being advanced. This review discusses the potential beneficial effects of probiotics in preventing and treating certain diseases as well as current and future perspectives of probiotic research.

Keywords: Probiotic; Heath benefits; Bile salt hydrolase; Dysbiosis; Microbiome

1. Introduction

Food fermentation and the consumption of fermented foods date far beyond human civilization. The transition from hunting and gathering to an agricultural lifestyle might have contributed to the further development of these food fermentations that are now practiced on industrial scales. However, human interactions with probiotics are more intimate and have a much longer history than the historic food fermentations. All parts of the human body such as the skin, oral cavity, gastrointestinal tract, and vaginal cavity are inhabited by trillions of microbes [1,2]. At birth, the human gut is sterile but colonized immediately after birth [3]. Factors such as the type of delivery (vaginal birth *versus* cesarean section) and the type of diet (breast feeding *versus* formula feeding) affect the colonization patterns [4]. The pioneer microbes that 'infest' the gut make permanent adaptations and determine the physiological, immune, metabolic and behavioral development and also influence future disease susceptibility [132]. Age and life style are some causes of many disease conditions since they contribute to alterations in the microbial flora in the body [5]. Recent studies have demonstrated that bacterial community composition is considerably altered in diseases such as obesity and periodontal disease, with healthy subjects usually exhibiting distinct, diverse and temporally stable bacterial populations at these sites when compared with patients displaying disease symptoms [6]. As consumers become aware of the impact of what they eat on their health, they tend to search for functional foods. Attention has been paid to prevention of diseases than cure and hence, probiotic containing foods are abundant on the market.

2. Diseases and disorders caused by alterations in the human gut microbiota

It is evident that prenatal maternal exposure influences postnatal microbial colonization [3] and this plays pivotal roles in gut-associated lymphoid tissue (GALT) development [7], specific aspects of immune system development [8,9] and the

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Table 1
Diseases and disorders caused by alterations in the gut microbiome.

Disease/disorder	Potential role of the microbiome	Recent findings
Atopy and asthma	• Pre- and postnatal microbial exposures influence immune development [3]. • Mode of delivery and nutrient uptake influence GI community development and protection against subsequent atopic disease development [13].	• Infants born by cesarean section are more often colonized with *Staphylococcus*, *Corynebacterium* and *Propionibacterium* and less with bifidobacteria and lactobacilli while vaginally delivered infants are colonized with *Lactobacillus*, *Prevotella* or *Sneathia* [14]. • *Streptococcus*, *Clostridium* species, *Bacillus subtilis*, *Bacteroides vulgatus* and *Veillonella parvula* are predominant in formula fed infants making them prone to allergic and autoimmune diseases [15].
Candida infection	• Depletion of gut microbiota permits *Candida albicans* proliferation and infection [131].	• Depletion of the gut microbiome through antibiotic administration is associated with increased *C. albicans* abundance and infection [131].
Celiac disease	• The GI of celiac disease patients contain large populations of Gram negative bacteria compared to healthy individuals [130].	• Pediatric celiac disease patients have significantly higher numbers of *Bacteroides*, *Staphylococcus*, *Salmonella*, *Shighella* and *Klebsiella* relative to healthy subjects [130]. • The ratio of *Lactobacillus–Bifidobacterium* species to *Bacteroides–E. coli* was lower for celiac disease patients [16].
Colorectal cancer	• High abundances *of Bacteroides* spp. and *Clostridium* spp. are present in the GI of CC patents [17].	• Overall bacterial diversity increased for CC patients compared with healthy controls [18]. • Microbial butyrate production causes apoptosis of CC cells [18].
Type I diabetes	• Interaction between the gut community and innate immune system may be a predisposing factor for diabetes [19]. • The microbiome plays a role in the development of insulin resistance [20].	• Metabolic profiling reveals a contribution of gut microbiota to fatty liver phenotype in insulin-resistant mice [20]. • The intestinal microbiota interacts with environmental factors and susceptible genetic factors, contributing to the development of diabetes [21].
Type II diabetes	• Gut microbiome dysbiosis is critical for pathogenesis [22].	• Low levels of *Roseburia intestinalis* and *Faecalibacterium prausnitzii* in the microbiome of Type II diabetics [22]. • Type II diabetes and obesity are highly influenced by gut microbiome [23]. • Gut microbiota may contribute to insulin sensitivity and cause low-grade systemic inflammation [20].
HIV	• Gut microbiome dysbiosis may be critical for pathogenesis [24].	• An important relationship exists between altered mucosal bacterial communities and intestinal inflammation during chronic HIV-1 infection [25]. • HIV-1-infected subjects had increased abundances of Proteobacteria and decreased abundances of Firmicutes compared with uninfected donors [24].
IBD	• Composition of gut microbiota contributes to inflammation [3]. • Treg-promoting organisms are depleted; overgrowth of bacteria that induce proinflammatory Th17 cell populations [26].	**Crohn's disease (IBDC)** • IBDC patients have high levels of Enterobacteriaceae, Pasteurellacaea, Veillonellaceae, and Fusobacteriaceae, and decreased abundance in Erysipelotrichales, Bacteroidales, and Clostridiales [27]. • IBDC patients have abnormal increase in antimicrobial dual oxidase (DUOX2) expression with increasing numbers of proteobacteria [28] • Fecal samples of CD patients have increased levels of *Bacteroides fragilis* (*B. fragilis*) reletive to control samples [129]. • An overall decrease in microbial diversity is observed in CD patients [128]. • CD patients have significant alterations in oxidative stress pathways, as well as decreased carbohydrate metabolism and amino acid biosynthesis in favor of nutrient transport and uptake [128]. • CD patients have leucine, isoleucine, valine, lysine, alanine, tyrosine, phenylalanine, glycine, glutamate, and aspartate malabsorption [29]. • Microbial diversity lower when compared with healthy individuals [29]. **Ulcerative colitis (IBDU)** • Lower levels of Bifidobacteria and *Clostridium leptum* [30] reported relative to healthy individuals. • TRUC gut microbiomes with active colitis has a reduced potential for both carbohydrate and energy metabolism and an enhanced potential for flagellar assembly, tetrathionate respiration and benzoate degradation [31].
IBS	• Disturbances of mucosa-associated bacteria may be important in the pathogenesis of IBS symptoms [32].	• Abnormal detection of hydrogen and methane in patients' breath suggests changes in bacterial fermentation [33]. • In children, a fecal microbiome with increased percentage of *Haemophilus parainfluenzae as well as* bacterial taxa from the genus *Alistipes characterizes IBS* [32].
Gastroenteritis	• Pathogenic species capitalize on GI microbial community disruption to elicit infection [34].	• *Helicobacter pylori* capitalizes on host disruption of GI microbiome to induce persistent inflammatory infiltration and can cause gastropathy and cancer [35].