Gastro-intestinal tract microbiota and human health.

Osman Erkmen*

Department of Food Engineering, Faculty of Engineering, University of Gaziantep, 27310 Gaziantep, Turkey

Opinion Article

The gastrointestinal tract (GIT) lies from the mouth to the anus. It is responsible with consumption and digestion of foods, absorption of nutrients, elimination of wastes, and immunological and physical barrier against toxic substances and pathogenic microorganisms. The organs including the stomach and small and large intestines are known as gut and microorganisms densely present in it. GIT microbiota contains about 1014 microorganisms and most of the microorganisms are bacteria [1]. The microbial number, type and functions vary in the human body but the majority (70%) of them present in the large intestine. Bifidobacterium, Ruminococcus, Lactobacillus, Streptococcus, Escherichia, Bacteroides and Clostridium are common genera of bacteria in the GIT of adults. About 60% of the bacteria is Bacteroides [1,2].

Microbiota in the GIT plays an important role in the metabolism, nutrition, physical protection, immunity and different processes outside of the GIT. Microbiota educates our immune systems early in life against foreign and harmful substances. They can stimulate host to secrete anti-inflammatory immune molecules to prevent over-activation of immune systems. Butyrate-producer Faecalibacterium prausnitzii produces anti-inflammatory compounds [1].

Gut (include stomach and intestines) microbiota responsible with the nutrient absorption and metabolism. They help to regenerate intestinal villi cells, which can increase surface area and nutrient absorption. They increase the peristaltic activity of the gut, which results with increasing movement of intestinal content down the gut. They produce different metabolic products [2]. Some of these metabolic products (such as butyrate and acetate) are short chain fatty acids which are used as nutrients by human cells (such as intestinal cells) and drive away free radicals and toxins. Butyrate can be used as nutrients by large intestinal cells, stabilize the tight junctions between cells, and prevent tumor formation. Acetate interacts with receptors in the human body and regulates storage of fat, utilization of glucose and regulates appetite. A protective mucus layer lines the intestinal cells which forms a protective barrier against pathogens may be stimulated by microbiota. They will then colonize through mucus layer, compete with pathogens for attachment sites and reduce available space on the intestinal surface against harmful microorganisms [1,3,4].

Fiber and protein utilization by microbiota in the large intestine produces physiologically important metabolic products, including short chain fatty acids which can be used by intestinal cells and bacteria, and influence inflammation and function of immune responses in tissues [5]. Gut microbiota produce vitamins (such as K, B12, biotin, folate, thiamine), acetate, lactate and amino acids [1,2]. Some microbiota (such as Lactobacillus, Bifidobacterium and Bacteroides) synthesis bile acids, which are important in lipid transport [6]. Microbiota in large intestine ferment undigested foods, especially carbohydrates/fiber, which can cause beneficial effect on the gut. Gut enzymes from microbiota influence food digestion, such as Bacteroides thetaiotamicron produces carbohydrate hydrolyzing enzymes [7]. Bacteria produce enzymes phytases in the large intestine and these enzymes degrade phytic acids which are present in grains. Phytic acid present as a complex with minerals (such as calcium, magnesium and phosphate). Hydrolysis of phytic acid in the intestines releases these minerals [8]. Enzymes degrade mucus and this is help to meet bacteria with nutrients and assists the mucus barrier lining the gut.

Many activities take place outside of the GIT. The gut-brain interactions are affected by microbiota and influence the human responses to stress. Changes in the microbial populations in the microbiota affect the central nervous system and brain activity [9]. Microbial products (such as acetate) can interact with the brain that helps to control appetite. Chronic stress can cause reduction of population in microbiota, and increases colonization and growth of pathogenic microorganisms in the GIT. Stressed people eat less healthful foods and consume more refined foods containing high fat and sugar. This type of foods negatively affects microbiota of GIT. Minimizing stress can have a healthful effect on microbiota. Setting aside time for relaxation, exercising regularly, and getting adequate sleep are just a few ways to prevent stress.

The GIT microbiota retains mainly affected by a long term dietary habits. High fiber diet from plenty foods supports health of gut. Short-term changes in diet can cause rapid and transient changes in microbiota. Choosing high fiber plenty foods (such as grains, fruits, vegetables and legumes) are the best ways to retain and support gut microbiota, and health of people. Dietary fiber consumption reduces the colorectal cancer risk with dilution and elimination of toxins through gut content, and increases metabolic activity of fermentative microbiota [10,11].

On the other hand, antibiotic use can eliminate some of the microbiota together with the targeted pathogenic bacteria from the GIT. After antibiotic use, functional foods containing probiotics and prebiotics or a probiotic supplement can help to restore the gut microbiota [12]. After the use of antibiotics, the original balance of gut microbiota may return within two weeks. Fermentation of protein by microbiota of large intestine after depletion of carbohydrates produces different toxic metabolites (such as ammonia, phenols, indoles, and hydrogen sulphide). These metabolic products have an important role in the large intestinal diseases including colorectal cancer and ulcerative colitis. Some biological active lipids, including Gram-negative bacterial lipopolysaccharide, can cause tissue
inflammation [2,13]. Many enteropathogenic bacteria can produce toxins to cause diarrhea [2]. Methanogenic Archaea (not bacteria) uses hydrogen to produce methane in the gut which may limit acetate and butyrate production by another microbiota [14]. Fermentation of nutrients by non-gut microbiota produces gases. Gut problems including bloating and pain can result from excess gas production [15]. Smoking induces changes microbial populations in the GIT that may increase risk of disease. Obesity can change gut microbial populations and mainly decrease number of Bacteroidetes [1]. Dietary saturated fat stimulates the formation of conjugated bile acids, and promotes growth of pathogens. Geography can also affect types of gut microbiota. The number of beneficial microbiota in children from rural areas would be greater than children from cities, since different microbiota associate with breakdown of fiber and dietary differences significantly changes type of microorganisms [16]. The type of fecal bacteria and their functional genes can also be different between individuals in the city and in rural areas [17]. Risk of infectious disease increases with travel, since this will also associate with changes in dietary foods. Poor hygienic conditions can change microbiota population in gut and increase spread of pathogenic microorganisms [2].

Every individual has a specific microbial species in GIT microbiota. Differences in GIT microbiota population among individuals may result from changes in utilization of dietary components. This can change susceptibility of individual to disease. Different factors (such as diet, infections, inflammation, physical protection, immunity, different processes outside GIT, and antibiotics use) can changes GIT microbiota. A diet with low fiber, and high fat and refined carbohydrates can cause microbial instability within the body (dysbiosis). Additionally, dietary changes, stress, infections, chronic diseases and pathogens can cause imbalance in the GIT microbiota. There are close associations between microbial instability of the GIT microbiota and disorders on the health of people.

References


*Correspondence to:
Osman Erkmen
Department of Food Engineering
Faculty of Engineering
University of Gaziantep
27310 Gaziantep, Turkey
E-mail: erkmen@gantep.edu.tr