

# Health and Disease: The Human Gut Microbiome

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## SHORT COMMUNICATION

The number of bacterial cells in the human Gastrointestinal Tract (GIT) outnumbers the number of cells in the host by a factor of ten, and the genes encoded by the bacteria in the GIT exceeds the genes in the host by more than 100 times. The gut microbiome refers to the bacteria that live in the human digestive tract. Extensive study has been conducted on the human gut microbiome and its significance in health and disease, demonstrating its importance in human metabolism, nutrition, physiology, and immunological function. Inflammatory Bowel Disease (IBD) and Irritable Bowel Syndrome (IBS) have been related to an imbalance of the normal gut microbiota, as well as larger systemic manifestations of disease such as obesity, type 2 diabetes, and atopy [1]. The stringent anaerobes exceed the facultative anaerobes (organisms that may thrive both aerobically and anaerobically) and aerobes in the gut microbiota by up to 100-fold. Despite the fact that more than 50 bacterial phyla have been identified in the human gut, the microbiota is dominated by only two: Bacteroidetes and Firmicutes. The number of bacterial species found in the human gut varies greatly between investigations, although it is usually assumed that each person carries more than 1000 species-level phylotypes. Because the gut microbiota encodes a significantly larger number of genes than its human host, it is able to perform a variety of metabolic processes that humans are unable to do or can only do to a limited extent. Gut bacteria may create a wide range of vitamins, synthesis all necessary and non-essential amino acids, and perform bile biotransformation. Furthermore, the microbiome provides essential biochemical pathways for the metabolism of nondigestible carbohydrates, such as resistant starches, cellulose, hemicellulose, pectins, and gums; some oligosaccharides that escape digestion; unabsorbed sugars and alcohols from the diet; and host-derived mucins. This feature allows the host to recover energy and absorbable substrates while also providing energy and nutrients to the bacteria for growth and proliferation. Carbohydrate metabolism is a primary source of energy in the colon. Many gut bacteria create antimicrobial chemicals and compete for resources and attachment sites in the gut lining, inhibiting pathogen invasion. The barrier or competitive-exclusion effect is the name given to this action [2]. Pathogenic bacteria can penetrate epithelial cells through attachment sites on host cells in the intestinal wall. Nonpathogenic bacteria can be found competing for these attachment sites on the border

of intestinal epithelial cells in laboratory research, preventing pathogenic, enteroinvasive bacteria from attaching and entering the epithelial cells. Furthermore, because bacteria compete for nutrients in their immediate environs and maintain their collective habitat by administering and consuming all resources, pathogenic bacteria can be outcompeted for resources by the intestinal microbiota by sheer numbers. Furthermore, bacteria can impede their competitors' growth by creating antimicrobial compounds known as bacteriocins, and the ability to synthesise these bacteriocins is widespread among gastrointestinal bacteria. The principal interface between the immune system and the external environment is the gut epithelium. Continuous and dynamic interactions with the gut microbiota and its metabolites influence the development of a host's immune system. Bacteria are essential to the gut-mucosal immune system's early development, both in terms of physical components and function, and they continue to play a role in its operation later in life. The cells of the intestinal epithelium ward off pathogens by signalling to the innate immune system via specific receptors that recognise and bind to specific molecules associated with bacteria, triggering the immune response and the release of protective peptides, cytokines, and white blood cells in the host. The end outcome could be a protective response to commensal bacteria, an inflammatory reaction to dangerous species, or a trigger for cell death in the host. Intestinal bacteria exposure has also been linked to allergy avoidance (i.e., a disproportionate reaction of the immune system to nonharmful antigens). Allergic newborns and early children have a distinct gut bacterial makeup than those who do not develop allergies. The gut microbiota is thought to boost the immune system and train it to respond proportionally to all antigens. Early life changes in the composition of the gut microbiota can result in an immune system that isn't well-trained and overreacts to antigens. Human gut microbiota has been linked to an ever-increasing range of illnesses, syndromes, and functional abnormalities. Anecdotal reports from people to data from major cohort studies all support these correlations. The following section summarises the most often discussed associations: the possibility of a link between the intestinal microbiota and (1) chronic gastrointestinal disorders like IBS and IBD, and (2) systemic metabolic diseases like type 2 diabetes and obesity [3].

Human gut microbiota evolves throughout time and appears to

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have a key role in both health and disease. The gut microbiota has a variety of beneficial roles in a healthy state, including energy recovery through the metabolism of nondigestible food components, protection from pathogenic invasion, and immune system modulation. Although the specific contribution of the gut microbiota to these diseases is unclear, a dysbiotic state of the gut microbiota is becoming recognised as an environmental factor that interacts with a host's metabolism and plays a role in pathological conditions, both systemic-obesity, diabetes, and atopy-and gut-related IBS and IBD.

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