JETIR.ORG ISSN: 2349-5162 | ESTD Year : 2014 | Monthly Issue
JOURNAL OF EMERGING TECHNOLOGIES AND INNOVATIVE RESEARCH (JETIR)
An International Scholarly Open Access, Peer-reviewed, Refereed Journal

THE ROLE OF MICROBIOME IN HEALTH AND DISEASE-A REVIEW

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Abstract*:* Our bodies are home to trillions of microorganisms. They play a vital role in various biological functions. The skin microbiome acts as the first line of defence against bacterial attack. Bacteria's are one of the main group of human skin microbiota. Chemical ingredients in cosmetics and preservatives used to prevent microbial contamination of products during their manufacture, use, or lifetime can cause changes in the composition of the skin's microbial flora. Other factors that influence skin health are pH, moisture present in the skin, and transepidermal water loss. The gut microbiota changes with several factors, including age, diet, and antibiotic use. After exposure, microbes can rearrange to maintain that balance, but several factors can leave a permanent mark on the gut flora: prebiotics, probiotics, fecal transplants, etc. Several treatment options already exist for, but their clinical applications are limited and restricted. Therefore, there remains a need to study the characteristic associations between the microbiome and gut microbiota-associated diseases. This helps in disease management and the development of diagnostic and monitoring tools. This review aims to discuss our gut microbiome and its relevance to human health and disease.

Index Terms **- Bacteria's, Skin microbiome, Prebiotics, Microbiota.**

Introduction-

Human skin biodiversity serves as an indicator of skin health. Several mechanisms have been shown to contribute to disease development when gut microbial homeostasis is disrupted. To prevent disease outbreaks, a balance must be maintained between defensive and pathogenic microbes. With a better understanding of this relationship, we explore possible ways to modify the gut microbiota as a backdrop for developing therapeutic options. , the surface microbiome opens up a new dimension. The host-specific microbiome forms microbial communities that have specific interactions with each other and with the host's immune system. Host neuroendocrine and immune pathways play an important role in establishing communication in the skin-gut-brain axis between commensal microbes and host internal organs. The human body is probably composed of 40% human cells and about 60% bacterial organisms. This fact raises more serious interest in how these microbes interact with specific hosts to influence host regulatory functions. These microbiomes are incredibly dynamic. They fluctuate constantly around a set point and vary from site to site. Microbiome composition is also determined by several environmental and local factors. For example, the pH and substrate of an organ site determine which microbes are suitable for survival and communion. Among the diverse microbiomes found in various human body sites, the gastrointestinal tract has the most abundant microbial community. Some are beneficial and some are pathogenic.

Origins of our gut microbiome

We have always believed that people are born with a sterile gut, until recent conflicting evidence supporting microbial transmission in the womb. I can do a lot of research. Studies of meconium microbiota have led to the understanding that intestinal colonization occurs before birth. This is demonstrated by the similarity of the meconium microbiota of neonates even when delivered by different methods (vaginal versus caesarean section). The endometrium and placenta are also not completely sterile. They may be a potential source of fatal intestinal colonization. However, it is still unclear exactly when and how colonization of the fatal intestine occurs. On the other hand, ingestion of amniotic fluid has been suggested as one possible method. At the birth of the infant, this process allows the microbiome to settle. Controversy therefore arises as to whether the route of administration can affect the human microbiome. New-borns delivered by caesarean section rather than vaginally have been observed to lack maternal vaginal and fecal bacterial (e.g., Lactobacillus and Prevotella) composition. However, recent studies have shown that the effect of the dosing regimen wears off by the second month of life. Other confounding factors may cause inconsistencies in gut microbiota between vaginal and caesarean infants. For example, maternal comorbidities are associated with caesarean deliveries and lower rates of exclusive breastfeeding in this group of mothers. It is interesting to know that breastfed infants up to 6 months of age have less diversity of intestinal flora than those who are not exclusively breastfed.

GUT MICROBES AND DISEASES

Galenism was once believed to explain disease caused by body fluids or fluid imbalances. It was after But this exciting concept lay dormant for decades until Louis Pasteur and Robert Koch successfully demonstrated the validity of germ theory. They have proven that microorganisms can cause disease. Scientists have observed symbiotic relationships between host microbial systems in the fecal

and oral microbiota. Additionally, environmental microbes have been observed to live in the communities involved. Human microbes are microbial communities that interact very delicately with each other and with their hosts, and their disruption can lead to a variety of health and psychiatric disorders. Much research is being done on the relationship between human microbes and many diseases. These studies have revealed the relationship between gut microbiota and human health.

1. Gastrointestinal diseases

1.1. Inflammatory bowel diseases

Inflammatory bowel disease (IBD) is a chronic inflammation of the gastrointestinal tract consisting of ulcerative colitis and Crohn's disease. The proinflammatory state of IBD may result from the host's inflammatory response to the gut microbiota in genetically susceptible individuals. Environmental factors contribute to disease development and recurrence. Comparing the individual's gut microbiota with his IBD and healthy counterparts, there are significant differences between them. IBD patients have been observed to have higher populations of pro-inflammatory microorganisms (Proteobacteria, Escherichia, and Fusobacterial) and lower populations of protective microorganisms (Bacteroidetes, Firmicutes, butyrate-producing genera). It is not surprising that individuals with IBD have lower levels of short-chain fatty acids (SCFAs) due to a reduction in butyrate-producing species responsible for the production of these molecules. The significance of the absence of these molecules and the reduced population of defensive microbes is that they have been shown to play an anti-inflammatory role in the immune response. Deficiencies in these microbes can lead to a proinflammatory state similar to the disease state of IBD patients. When discussing differences in the gut microbiota of IBD patients and healthy individuals, it is not certain whether this is a cause or a consequence of the disease. We were able to show that genetic factors such as domain-containing protein 9 (CARD 9), and autophagy-associated 16-like 1 (ATG16L1) are responsible. Difference. Expression of these genes is responsible for their anti-inflammatory and antibacterial effects in the gastrointestinal tract. Moreover, they also help regulate the homeostasis of the gut microbiota by determining its composition. Has been shown to be susceptible to colonization by On the other hand, ATG16L1 mice were observed to have a higher population of Bacteroidetes species with anti-inflammatory roles. Altering this gene expression may therefore disrupt intestinal colonization and its inflammatory response and defence against pathogens. Extrapolating this to the human host explains the susceptibility of genetically susceptible individuals to disruption of intestinal homeostasis leading to inflammatory bowel disease.

The terminal ileum is the most affected intestinal segment in Crohn's disease and the colonic segment in ulcerative colitis. This corresponds to sites in the gastrointestinal tract of IBD patients with higher concentrations of microorganisms than healthy individuals. Parallel distribution.

1.2. Colorectal cancer

Colon cancer is one of the most common types of cancer affecting both men and women. Chemotherapy resistance is a well-known hurdle for colorectal cancer treatment. Therefore, there is interest in investigating the anticancer potential of novel actinomycetes as effective treatments for colon cancer. Recently, studies have revealed the role of colonic microbes in the carcinogenesis of colorectal cancer. Evidence from these studies is supported by a positive correlation between microbial concentration and cancer cell distribution. Cancer is associated with our immunological and genetic dysregulation. Gut microbes are known to be involved in carcinogenesis by inducing inflammatory and carcinogenic states in the gut. Some microorganisms act as 'bacterial drivers' that damage DNA in the intestinal epithelium. This can result in hyper proliferation of epithelial cells, the ability of cells to evade apoptosis, etc., which are hallmarks of cancer. In addition, 'bacterial drivers' can disrupt gut integrity and promote the growth of pathogenic 'passenger microbes' that have a synergistic effect with 'bacterial drivers'. Wu et al. have demonstrated specific intestinal commensal mechanisms that activate the host's immune system, thereby creating a pro-inflammatory state favourable to cancer development. It can be hypothesized that intestinal homeostasis is disrupted and pathogenic commensals overgrow, leading to the development of colon cancer.

1.3. Gastric cancer

H. pylori affects approximately 50% to 60% of the world's population. It is known to cause stomach cancer in 1-3% of people. With the advent of Louis Pasteur and Robert Koch's germ theory, research on the role of the microbiome in disease has flourished. In the late 19th century, a relationship between gastric microbes and gastric cancer was proposed, but the dominant organism of gastric microbiota has not yet been identified. This was later disputed by Heinemann and Ecker, who observed in previous studies that the proposed microorganism was Lactobacillus. Decreased stomach acid allows this species to grow and is not the cause of stomach cancer. After the discovery of Helicobacter pylori in the late 19th century, researchers began to uncover the relationship between the gut microbiome and stomach cancer. .

It is associated with H. pylori infection and contributes to about three-quarters of all cases of gastric cancer. They are multiple strains of H. H. pylori each have distinct virulence factors that can cause genetic mutations that affect cell division, function, and immune response of the gastric epithelium.

One proposed mechanism is that Helicobacter pylori generates reactive oxygen species, causing DNA mutations, methylation defects, and oxidative damage, which may lead to loss of functioning tumour suppressor genes. That's what it means. Another virulence factor worth mentioning is Cag-A.

Protein. Cag-A is associated with an increased risk of premalignant and malignant gastric lesions. H. pylori uses its adhesion molecules for long-term colonization of the gastric epithelium and injects oncogenic proteins, including Cag-A, into gastric epithelial cells. This oncogenic protein causes changes in epithelial cell morphology and disrupts epithelial intercellular junctions. In addition, these molecules cause metaplasia and dysplasia of the epithelial layer, leading to the development of gastric cancer.

H. pylori has a vac-A toxin that disrupts the mitochondrial membrane, creates vacuoles in the cytoplasm, and causes apoptosis of the gastric epithelium. This is consistent with the mechanism of atrophic gastritis caused by H. pylori, which subsequently undergoes epithelial metaplasia and dysplasia, leading to the development of gastric cancer. Moreover, these virulence factors promote inflammation and create a favourable environment for tumorigenesis. Destroyed intestinal epithelial cells begin to proliferate uncontrollably, leading to stomach cancer. Saenz et al. suggested that the gastric epithelium may respond to injury through cellular plasticity and reprogramming. However, the mechanism.

2. Metabolic disorders

2.1. Obesity

Microorganisms play many potential roles in our gut, from digestion to regulation of absorption to metabolism of drugs and substances. They can also synthesize molecules such as short-chain fatty acids that affect the immune and metabolic systems. Not surprisingly, obesity is part of a metabolic disorder. Disturbances in intestinal homeostasis can impair the balance between energy intake from food and its expenditure. This can be explained by the role of microorganisms in processing nutrients. It can break down indigestible foods and convert them into short-chain fatty acids (SCFAs) such as acetate, butyrate, and propionate. These substances are involved in different functions and activities in different organs. Butyrate, acetate, and propionate are required for the production of glucose and lipids in the liver. They stimulate enter endocrine L cells to release glucagon-like neuropeptide 1 and local factor peptide YY, thereby regulating lipid digestion and metabolism in addition to fatty acid deposition in the liver. At the same time, butyrate also serves as an important energy source for colonic epithelial cells. The concentrations of these end-products depend on the host's gut microbiota. Different gut microbiota are associated with different metabolic properties. Turn bow etc. Genetically obese mice have been shown to have higher levels of acetate and butyrate in the gastrointestinal tract and lower energy levels in their faeces than genetically lean mice. This indicates that the intestinal community of genetically obese mice is better at producing energy than lean mice. For example, butyrate and propionate have been shown to increase satiety.

2.2. Type 2 diabetes

Type 2 diabetes (T2DM) is a chronic disease characterized by elevated blood sugar levels, relative insulin deficiency, and insulin resistance. It is also associated with other metabolic abnormalities such as Incretin deficiency, elevated glucagon levels, and increased lipolysis. Like other chronic diseases, type 2 diabetes has been attributed to an abnormal intestinal environment. This was reported by Buckhead et al. Even when mice have similar genotypes and controlled dietary patterns, different gut microbiota show different profiles of glucose metabolism. This indicates that diverse gut microbiota with different capacity for glucose metabolism and low glucose metabolism may predispose the host to T2DM. It has also long been known that T2DM is associated with mild systemic inflammation leading to host insulin resistance. This pro-inflammatory condition may result from the host microbiome and may play a role in metabolism and immunology. As in other chronic diseases, reduced gut microbial diversity has been observed in patients with type 2 diabetes. This allows pathogenic microbial overgrowth to promote local inflammation by activating innate immunity.

2.3. Pulmonary health and asthma

Until recently, the lower respiratory tract was considered sterile. Studies have found microbes in the lungs of even healthy people. Pulmonary microbes are detectable shortly after birth and their composition is enriched by upper respiratory and oropharyngeal microbes. The concept of the gut-lung axis has also been proposed in parallel with the growing interest in correlations between human microbes and disease. This was suggested by his Schuijt et al. demonstrated that mice depleted of gut microbes exhibited more common disease, complications and mortality when infected with pneumococci. Transplantation of fecal microbes into pneumococcal-infected mice depleted of gut microbes reduced the pulmonary inflammatory response. This indicates that the gut microbiota can enhance host defences against pulmonary infections. Asthma is an atopic airway disease characterized by chronic inflammation of the small airways. Comparing the microbiome of asthmatics with that of healthy individuals shows that asthmatics have a higher number and diversity of microbes. Colonization of the infant's oropharynx with Moraxella, Haemophilus, and Streptococcus in the first year of life has been shown to predict the risk of developing asthma later in life. One study comparing gut microbiota showed that the population of faecal bacterium, Lachnospira, Villanelle, and Rothia genera in infants at high risk for asthma decreased at 100 days of age. On the other hand, another study showed an increase in Clostridium neonaterre and Lachnospira in the gut microbiota of infants. This indicates the risk of asthma in preschool age.

3. Mental health

The concept of the gut-brain axis can be traced back to the early 20th century, where the functional integrity of the gut and microbes has been recognized as part of the pathophysiology of mental health disorders. Researchers at the time believed that self-intoxication could impair mental health. They suggested that toxic intestinal contents could have serious effects. In recent years, a growing body of research supports this bidirectional relationship between the brain and the gut. Gut microbes produce metabolites and molecules that interact with the brain and can regulate the immune system. Conversely, the brain can alter the composition of the gut microbiota via neural signals. Gut microbiota homeostasis has been shown to protect the integrity of the gut and blood-brain barrier. If this balance is disturbed, pathogenic microbes can overgrow and cause local inflammation. It then increases intestinal permeability, allowing intestinal contents to enter the systemic circulation and triggering a systemic immune response. Proinflammatory conditions have been shown to alter the integrity of the blood-brain barrier. This allows Immunomodulators and pathogenic microbes to enter the brain. On the other hand, some gut microbes produce metabolites that indirectly alter brain function through neural pathways. In short, this shows that an imbalance in the gut ecosystem can impair brain function, making people more susceptible to developing mental health disorders. , pro-inflammatory conditions have been observed in individuals with mental health disorders. Higher activated c1q levels are observed in individuals with schizophrenia, and increased intestinal permeability and inflammatory status are observed in individuals with autism spectrum disorders.

The human gut microbiota evolves throughout life and appears to play an important role in both health and disease. It has a myriad of beneficial functions, including protecting the host from pathogen invasion and regulating the immune system. An abnormal state of the gut microbiota interacts with the host's metabolism and is implicated as an environmental factor that plays a role in both systemic (obesity, diabetes, atopy) and gut-related his IBS and IBD morbidity. Increasingly recognized. The gut microbiome for these diseases is unknown. Heterogeneous etiologies of metabolic and gastrointestinal diseases are associated with different microbes, but little information is available on the causal relationships of associations.

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