World Journal of Biology Pharmacy and Health Sciences
eISSN: 2582-5542
Cross Ref DOI: 10.30574/wjbphs
Journal homepage: https://wjbphs.com/

How do different bacteria affect the gut?

Aryan Hanji *

IBDP 2-year student, Jay Shree peril international school, Jaipur, Rajasthan, India.

World Journal of Biology Pharmacy and Health Sciences, 2025, 22(02), 472–479

Publication history: Received on 13 March 2025; revised on 22 April 2025; accepted on 24 April 2025

Article DOI: https://doi.org/10.30574/wjbphs.2025.22.2.0409

Abstract

This research paper highlights the different varieties of bacteria that affect the human gut and how it influences various bodily systems, such as the cardiovascular, nervous, and immune systems. This study reflects microflora's role in digestion, absorption of nutrients, and prevention from some diseases. The dietary habits and their impact on gut microbiota diversity on certain conditions, such as coronary heart disease (CHD), non-alcoholic fatty liver disease (NAFLD), and cancers, are specifically focused on. The interaction of gut bacteria alongside inflammation, respiratory health, and oral hygiene was also examined. The findings reiterate the vital necessity of a balanced gut microbiome for human health and prevention from chronic diseases

Keywords: Bacteria; Gut; Cardiovascular; nervous, and immune systems; Diseases

1. Introduction

1.1. What are microbes? How do they Enter the human body?

Microbes are tiny living things that are found all around us. They are also referred to as microorganisms and are too small to be seen with the naked eye. They live in water, soil, and the atmosphere. Millions of these microbes live in the human body, as well. (1). Some microbes make us ill, others are important for our health. The most common types are bacteria, viruses, and fungi. There are also microbes called protozoa. These are tiny living things that are responsible for diseases such as toxoplasmosis and malaria. (1). This research paper mainly focuses on bacteria. Bacteria are tiny living organisms consisting of a single cell. The term for a single bacterium is "bacterium." Countless types of bacteria are present worldwide, they can be found in various locations, including within the human body. They inhabit the skin, airways, and mouth, as well as the digestive system, reproductive system, and urinary tract. Such bacteria are called resident flora. According to scientists, the human body contains approximately 10 times more bacterial cells than human cells. (2).

Microbes usually enter our bodies through the mouth, eyes, nose, or urogenital openings, or through wounds or bites that breach the skin barrier.(5)

1.1.1. Microflora

MicroFlora is the term for bacteria and other organisms that reside in the intestines. They aid in food digestion. Microflora produces vitamins, including K and biotin. Also known as intestinal flora, intestine microflora, gut flora, and gut microflora. With more than 400 different bacterial species, the intestinal microflora is a complex ecology. There are more anaerobes than facultative anaerobes. In the lower colon, the flora is abundant, while in the stomach and upper intestine, it is scarce. Although they are found in the lumen and adhered to the mucosa, bacteria often do not pass through the intestinal wall. (3). The enterohepatic circulation which is the movement of bile acid molecules from the

* Corresponding author: Aryan Hanji

liver to the small intestine and back to the liver, relies on intestinal bacteria to deconjugate metabolites that are conjugated in the liver and expelled in the bile. The bacterial enzymes in the gut then absorb the metabolites via the mucosa and return them to the liver through the portal circulation. The circulation of many medications and endogenous substances occurs in the liver. The fecal excretion and, consequently, the blood levels of these chemicals can be changed by antibiotics that restrict the flora. Additionally, the flora synthesizes several vitamins and aids *in the digestion of fiber.(4).*

1.2. What is the food habit that is affecting the gut?

In addition to giving our bodies the essential nutrients they require, food components in our diet serve as substrates for the mutualistic microbial flora in our gastrointestinal tract, known as the gut microbiome. Food ingredients that are not fully digested are *converted into a wide variety of* metabolites. In order to maintain intestinal homeostasis in a healthy condition and interact with the gut epithelium and mucosal immune system, the form, content, and function of the gut microbiome are shaped by the foods we eat. (6). The main nutrient for preserving the variety of gut bacteria is dietary fiber. Many chronic inflammatory conditions like obesity, diabetes, and IBD are linked to low diversity of gut bacteria. The primary cause of the depletion of fiber-degrading microbes in industrialized societies is a diet high in fat and *protein and low in fiber. (7).*

1.3. What is the diversity of bacteria should we have to avoid CHD?

High rates of morbidity and death are characteristics of cerebrovascular and cardiovascular disorders. Cardiovascular illness and microbiota are intimately related.(8).Cardio-cerebrovascular disease is thought to be either directly or indirectly influenced by the human gut flora.(9). Studies have shown a connection between the gut microbiota and coronary heart disease risk factors such as diabetes, obesity, dyslipidemia, and hypertension.(10). Atherosclerosis and coronary heart disease can arise as a result of the metabolites that the gut microbiota produces that are linked to the metabolism of cholesterol, uric acid, oxidative stress, and inflammation.(11). Research depicts that patients with high blood pressure (HBP) are associated with obvious gut microbiota disturbance and intestinal mu*cosal barrier dysfunction.(12).*

2. Cardiovascular diseases

Endocarditis is a life-threatening inflammation of the inner lining of the heart's chambers and valves. This lining is called the endocardium.

Endocarditis is usually caused by an infection. Bacteria, fungi or other germs get into the bloodstream and attach to damaged areas in the heart. Things that make you more likely to get endocarditis are artificial heart valves, damaged heart valves or other heart defects.Without quick treatment, endocarditis can damage or destroy the heart valves.

Treatments for endocarditis include medications and surgery. Endocarditis may develop slowly or suddenly and It depends on the type of germs causing the infection and whether there are other heart problems.

Common symptoms of endocarditis include aching joints and muscles, chest pain, fatigue, flu-like symptoms, night sweats, swelling in feet, etc. Usually, the body's immune system destroys any harmful bacteria that enter the bloodstream. However, bacteria on the skin or in the mouth, throat or gut (intestines) may enter the bloodstream and cause endocarditis under the right circumstances. Endocarditis occurs most often in adults over age 60(18). Different bacterial species related to the risk of CVD include Helicobacter pylori, Chlamydia pneumonia, Mycoplasma pneumonia an*d Porphyromonas gingivalis(19).*

3. The Immune System

The immune system, though intricate in design, functions to safeguard the body from invading pathogens through its vast army of cellular soldiers and specialized lymphatic structures. Defending host health is no easy feat, as disease-causing microbes *are in a perpetual arms race of* mutation. From the thymus gland training T cells to the spleen and bone marrow churning out immune cells, a carefully choreographed network of tissues and cellular agents patrols the bloodstream and tissues. When interlopers breach the body's perimeter, lymph nodes rapidly swing into action, summoning lymphocytes to remember and eliminate the microbial foe. While complexity brings vulnerability, constant evolutionary interplay has driven the immune system to ever more finely-tuned sentinels recognizing and responding to threats foreign to the body.Humans and other mammals live in a world that is heavily populated by both pathogenic and non-pathogenic microbes, and that contains a vast array of toxic or allergenic substances that threaten normal homeostasis. The community of microbes includes both obligate pathogens, and beneficial, commensal organisms, which the host must tolerate and hold in check in order to support normal tissue and organ function. Pathogenic microbes possess a diverse collection of mechanisms by which they replicate, spread and threaten normal host functions. At the same time that the immune system is eliminating pathological microbes and toxic or allergenic proteins, it must avoid responses that produce excessive damage of self-tissues or that might eliminate beneficial, commensal microbes. Our environment contains a huge range of pathogenic microbes and toxic substances that challenge the host by a very broad selection of pathogenic mechanisms. It is not surprising, therefore, that the immune system uses a complex array of protective mechanisms to control and usually eliminate these organisms and toxins. A general feature of the immune system is that these mechanis*ms rely on detecting structural* features of the pathogen or toxin that mark it as distinct from host cells. Such host-pathogen or host-toxin discrimination is essential to permit the host to eliminate the threat without damaging its own tissues.(13).

4. Gut against Bacteria

The stomach defends against harmful bacteria with two barriers: the mechanical barrier and the immune barrier. The physical obstruction is made up of one layer of polarized intestinal epithelial cells, specifically the enterocytes, along with mucus. In contrast, the immune barrier comprises secreted IgA, intraepithelial lymphocytes, macrophages, neutrophils, natural killer cells, Peyer's plaques, and mesenteric lymph nodes.

Beneficial bacteria and probiotics can support the health of gut barriers. Beneficial bacteria in the gut aid in protecting the host by preventing pathogenic invaders and supporting immune system growth. Gut bacteria prevent pathogenic bacteria colonization by competing for nutrients and attachment sites on the colon's mucosal surface, a process called "colonization resistance." Commensal bacteria(examples to include) reduce intestinal pH by producing lactate and short-chain fatty acids (SCFAs) to prevent the invasion of pathogenic bacteria . An alternative method involves the production of harmful or cancer-causing substances to prevent the growth or eliminate harmful bacteria, along with volatile fatty acids that can prevent the growth of harmful bacteria.(14).Gut bacteria not only support the host's gut defense system, but also assist in maintaining normal gut functions. The host is helped by gut bacteria in multiple ways like controlling gut movemen*ts, creating vitamins, changing* bile acid and steroids, breaking down foreign substances, taking in minerals, and triggering and eliminating toxins, genotoxins, and mutagens. The nearby part of the colon generates a large amount of short-chain organic acids, like acetic, propionic, and butyric acids. These organic acids provide energy for the lining of the colon and other tissues in the body. They are produced by the breakdown of complex carbohydrates through fermentation by bacteria in the colon. As a result, these natural acids influence the growth of bacteria in the colon by impacting water absorption and reducing the pH of feces (15).

4.1. How our nervous system is getting affect by bacteria?

Infections of the central nervous system (CNS) are among the most devastating infectious diseases worldwide and often result in medical emergencies that require prompt management. Pathogens may access the CNS by crossing the blood–brain barrier (BBB), which normally protects the CNS from microbial invasion, or via transneuronal routes that bypass the blood-brain barrier.

A brain abscess which is a pocket of pus may form when bacteria from an infection elsewhere in the head or in the bloodstream or from a wound enter the brain(16).

Foods that affecting our brain

A neurotransmitter called serotonin mediates moods, inhibits pain, and controls hunger and sleep.The billions of "good" bacteria that comprise your gut micro biome have a significant impact on how these neurons operate as well as how neurotransmitters like serotonin are produced. These microorganisms are vital to your well-being.

They enhance your ability to absorb nutrients from meals, reduce inflammation, protect the lining of your intestines and make sure they form a strong barrier against toxins and "bad" bacteria, and stimulate neuronal connections that go straight from the gut to the brain(17).

Various Bacteria such as Lactobacillus and Bifidobacterium help in producing the neurotransmitter serotonin. These

can be found in yogurt and sauerkraut.

4.2. Liver

In addition to transporting blood from the gastrointestinal tract to the liver, the portal vein can also transport bacteria. This suggests that a wide range of microbial illnesses may affect the liver. Certain infections primarily affect the immunodeficient host, whereas others have an impact on immunological competence. Hepatic infections can be primary or secondary, occurring as a component of infectious or systemic diseases.

There are two types of bacteria, gram positive and gram negative. Gram positive have thick call walls and gram negative bacteria have thin cell walls. These both bacteria release toxins and affect the different body parts. There are several gram-positive and gram- negative bacterial infections that may lead to hepatic compromise. Hepatic compromise refers to a state of impaired or reduced liver function. Hepatic expression of diseases includes appearance of jaundice, Yellowing of the skin, Itchy skin,etc. Progression of disease may lead to extensive hepatic necrosis(death of hepatocytes which are responsible for a variety of cellular functions in the liver) and liver failure.The main cause of hepatic compromise is Staphylococcus aureuswhich i*s a gram positive bacteria.(20)*

Nonalcoholic fatty liver disease (NAFLD) is a metabolic liver disease that can eventually lead to liver cirrhosis(a condition in which the liver is scarred and permanently damaged) and hepatocellular carcinoma(a common liver cancer caused by hepatitis B or hepatitis C infection). *Porphyromonas gingivalis* (P.g) is the main pathogen that causes dentistry disease, which participat*es in the development of NAFLD.*

In NAFLD, too much fat builds up in the liver. It is seen most often in people who are overweight or obese. NAFLD usually does not have symptoms but when it does, pain or discomfort in the upper right belly area or fatigue is observed. Treatment for NAFLD usually starts with weight loss. This can be done by eating a healthy diet, limiting portion sizes and exercise. Losing weight may improve other health prob*lems that lead to NAFLD(21).*

5. Bacteria affecting the chance of cancer

But in recent years, two methods have been used to connect bacteria to cancer: the creation of carcinogenic bacterial metabolites and the activation of chronic inflammation. An instance of the inflammatory mechanism of carcinogenesis(process by which normal cells are transformed into cancer cells) that is most particular is the Helicobacter pylori infection. H. pylori's tendency to generate chronic inflammation has led to epidemiological links between it and distal stomach cancer. Through the stimulation of cell proliferation and the generation of mutagenic free radicals this inflammation is hypothesized to be the origin of cancer(22).H. pylori enters the body through the mouth, moves through the digestive system, and infects the stomach or the first part of the small intestine. The spiral- shaped bacterium uses its tail-like flagella to move around and burrow into the stomach lin*ing, which causes inflammation.*

The most prevalent biliary tract cancer in the world, gall bladder cancer (GBC), has a very bad prognosis since it is typically discovered at an advanced stage because of its delayed clinical presentation and metastases to the liver, peritoneum, and regional lymph nodes.Studies have shown association of Salmonella typhi and Helicobacter pylori with GBC(23).S. typhi releases harmful substances during the long-term infection phase, even when there are no visible symptoms. These substances can help turn normal cells into cancer cells. An infection with S. typhi might be an important warning sign for developing gallbladder cancer (GBC) and can be very dangerous in free-floating conditions.

5.1. Respiratory problems associated with the Gut

Gastrointestinal disorders or symptoms frequently accompany respiratory viral infections and chronic respiratory conditions, such as asthma and chronic obstructive pulmonary disease (COPD).

Individuals who suffer from gast*rointestinal disorders, such as* gastroesophageal reflux disease and inflammatory bowel disease (IBD), are more likely to experience respiratory illnesses and pulmonary dysfunction. These links imply that the gut and lung communicate vitally.The gut microbiota is affected by many factors, such as drugs, diet, mode of delivery, and feeding practices, which may play a role in susceptibility to respiratory diseases(24).

Inflammatory reaction and ways good bacteria try to stop it and cure The immune system's reaction to infections, damaged cells, poisonous substances, or radiation is *inflammation, which eliminates* the damaging stimulus and starts the healing process. Thus, inflammation is an essential defensive mechanism for good health. Cellular and molecular processes and interactions often effectively reduce the risk of infection or harm during acute inflammatory reactions. This mitigation step helps to resolve the acute inflammation and restore tissue homeostasis. Unchecked acute inflammation, on the other hand, can develop into chronic inflammation and lead to a number of chronic inflammatory illnesses(25).Inflammation is characterized by redness, swelling, heat, pain, and loss of tissue function, which result from local immune, vascular and inflammatory cell responses to infection or injury.Various pathogenic factors, such as infec*tion, tissue injury, or cardiac* infarction, can induce inflammation by causing tissue damage. The etiologies of inflammation can b*e infectious or non-infectious.*

Suppressing the inflammatory response is necessary to stop further tissue damage and the evolution of acute inflammation into chronic, persistent inflammation. Chemokine gradients are gradually neutralized throughout the well-managed process of inflammation resolution, which involves the generation of mediators under temporal and geographical control. White blood cells in circulation gradually lose their ability to detect these gradients and are not drawn to damage sites. Chronic inflammation that is out of control can result from this process' dysregulation. Reduction or cessation of neutrophil tissue infiltration and apoptosis, counter-regulation of chemokines and cytokines, macrophage conversion from classically to alternatively activated cells, and the start of healing are examples of inflammation resolution processes that restore tissue homeostasis.

5.2. Oral problems associated with gut bacteria

Dental caries, periodontal disease, obesity, and cardiovascular disease are just a few of the local and systemic human disorders that have been linked to oral microbiom*e dysbiosis. The number of taxa* linked to pathogenic states can be decreased with the support of good oral hygiene practices. For instance, cleaning the teeth and tongue significantly reduces bacteria linked to dental illnesses, and flossing has been linked to lower concentrations of the dental pathogen Streptococcus mutans.By identifying bacterial shifts that took place when human cultures moved from a hunter-gatherer diet to a more carbohydrate-rich diet linked to farming, the impact of nutrition on oral microbiome composition was evaluated. However, the complete effects of the contemporary high carbohydrate and sucrose diet are still being determined. Diets associated with Western industrialized civilizations have been proven to cause poor oral health conditions, with high sugar content affecting bacterial relative abundances.

5.3. Respiratory problems associated with the gut bacteria

Chronic respiratory diseases, including asthma and chronic obstructive pulmonary disease (COPD), as well as respiratory virus infection, are often accompanied *by gastrointestinal diseases or* symptoms. Patients with gastrointestinal diseases, such as inflammatory bowel disease (IBD) and gastroesophageal reflux, are prone to develop pulmonary dysfunction and have an increased incidence of respiratory disease(26). The microbiota of the upper and lower respiratory tracts are distinct, with more Firmicutes and Actinobacteria in the nostril and more Firmicutes, Proteobacteria, and Bacteroidetes in the oropharynx , whereas there are more Bacteroidetes and Firmicutes in the lung . At the genus level, Bacteroides, Faecalibacterium, and Bifidobacterium are enriched in the gut , while Prevotella, Veillonella, and Streptococcus are the prominent genera in the lung . Although the gut and respiratory microbiota exhibit compositional differences, the epithelia of both the GI and respiratory tracts develop from a common embryonic structure, the anatomical structures and functions of the two mucosal sites are similar, and early-life microbial colonization of the gut and lung exhibits similarities. Therefore, accumulating evidence has highlighted the relationship and crosstalk between the gut and lung, referred to as the gut-lung axis.

Specific gut bacteria have also been found to be closely related to asthma. For instance, Clostridium and Eggerthella lenta were more abundant in the gut of asthma patients. Additionally, a child's risk of developing allergies and asthma increased when the abundances of Anaerostrips and Blautia increased and those of Faecalibacterium ,

Bifidobacterium and Fusicatenibacter decreased. Clostridiumproduce toxins or metabolites that can promote systemic inflammation, which might exacerbate or trigger asthma-like symptoms.SCFAs(Short-chain fatty acids), such as butyrate, are produced by Bifidobacterium and Faecalibacterium and are essential for their anti-inflammatory properties. A decrease in these bacteria may result in a decrease in these SCFAs, which could worsen lung inflammation.(27).

Table 1 All the bacteria and the food source

Bacteria	Primary location	Effects	Food source
Helicobacter pylori	Stomach (gastric mucosa)	Causes chronic gastritis and ulcers. Induces systemic inflammation, contributing to atherosclerosis.	Contaminated water, raw or undercooked vegetables.
Helicobacter pylori	Stomach (gastric mucosa)	Causes chronic gastritis and ulcers. Induces systemic inflammation, contributing to atherosclerosis.	Contaminated water, raw or undercooked vegetables.
Chlamydia pneumoniae	Respiratory tract	- Infects endothelial cells, leading to vascular inflammation and plaque formation in arteries (atherosclerosis)	Respiratory droplets; not directly foodborne.
Porphyromonas gingivalis	Oral cavity (dental plaque)	Causes gum disease (periodontitis). Translocates to systemic circulation, promoting atherosclerosis.	Found indirectly through poor oral hygiene; not foodborne.
Escherichia coli (E. coli)	Gut (colon, small intestine)	Produces metabolites that affect the gut-brain axis. Linked to neuroinflammation and conditions like anxiety.	Contaminated water, undercooked meat, raw vegetables.
Streptococcus spp.	Oral cavity, gut	Translocates to the brain, potentially causing meningitis or infections. Associated with neurodegenerative diseases.	Found in contaminated dairy products and foods stored improperly.
Lactobacillus spp.	Gut (small intestine, colon)	- Positively influences the brain by producing neurotransmitters (e.g., GABA) that regulate mood and anxiety.	Fermented foods like yogurt, kimchi, sauerkraut.
Porphyromonas gingivalis	Oral cavity, gut (via translocation)	- Contributes to non- alcoholic fatty liver disease (NAFLD) and inflammation in the liver.	Indirectly through poor oral hygiene, not foodborne
Staphylococcu s aureus	Skin, nasal passages	- Can cause liver abscesses if it spreads to the liver via the bloodstream. - Produces toxins leading to liver inflammation.	Contaminated dairy, meat, and improperly stored food.
Salmonella typhi	Gut (small intestine)	- Causes typhoid fever, which can lead to liver enlargement and hepatitis. - Can induce liver inflammation and dysfunction through systemic infection. -Can affect the chances of Gall bladder Cancer	Contaminated water, raw fruits, vegetables, and undercooked meat.
Helicobacter pylori	Stomach (gastric mucosa)	- Linked to liver diseases such as non-alcoholic fatty liver disease (NAFLD). - Causes systemic inflammation impacting liver health.	Contaminated water, raw or undercooked vegetables.
Clostridium	Gut (colon)	- Produces toxins leading to liver damage (e.g., Clostridium difficile can cause liver-related sepsis). - Promotes liver abscesses and inflammation through bacterial overgrowth or translocation.	Contaminated food, especially canned foods or improperly preserved items (e.g., meats, vegetables).
Eggerthella lenta	Gut (colon)	- Modulates bile acid metabolism, potentially affecting liver function. - Associated with liver inflammation in cases of dysbiosis.	Found naturally in gut microbiota; not directly foodborne. However, dysbiosis can be influenced by low-fiber diets.

5.4. Future Perspectives

Future research need to explore much neater how gut bacteria impact systemic disease. Further advancements in metagenomics and personalized medicine offer a sufficiently hopeful note of targeted therapies for restoration of a healthy microbiota. Probiotic and prebiotic interventions may also serve as non-invasive treatments for gut dysbiosis-associated conditions. Further longitudinal studies are needed to assess the long-term impact of dietary habits on gut health across populations. Further advancement into gut-brain axis interactions may also offer new avenues for managing nervous and psychological disorders. These could bring microbiome research into clinical practice and refocus future medical care on prevention through a virtuous cycle of exploitation of the body's very own microbe ecosystem.

6. Conclusion

In this review it is shown how the effects of various bacterial species on the human gut are multiple and how the gut microbiome influences an individual's overall health through pathways involving the cardiovascular system, immune response, nervous system, and the liver and lymphatic system, as well as respiratory pathways. The results present new evidence that having a balanced gut microbiome can help prevent chronic disease including items such as cardiovascular heart disease (CHD), non-alcoholic fatty liver disease (NAFLD), neuroinflammation, and certain cancers. Furthermore, the study presented illustrates the interconnections among the diet, personal hygiene, and microbial diversity in contributing to support or detract from whole-body homeostasis. By bringing together a contemporary body of work, this paper has presented a nuanced perspective of how gut bacteria can contribute to health and disease processes. It is hoped that this type of study will serve to further benefit society, by increasing awareness about the gut microbiome and disease prevention, as well as instigating dietary and lifestyle changes as a future method for the development of a healthier society.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

References

[1] National Institute of Allergy and Infectious Diseases (NIAID), 2009. Understanding microbes in sickness and in health.

[2] Bush, L.M. and Charles, E., 2019. Schmidt College of Medicine, Florida Atlantic University. Последний полный пересмотр/исправление июнь.

[3] Gorbach, S.L., 1971. Intestinal microflora. Gastroenterology, 60(6), pp.1110-1129.

[4] Gorbach, S.L., 1996. Microbiology of the gastrointestinal tract. Medical Microbiology. 4th edition.

[5] Drexler, M., 2014. What you need to know about infectious disease.

[6] Zhang, P., 2022. Influence of foods and nutrition on the gut microbiome and implications for intestinal health. International journal of molecular sciences, 23(17), p.9588.

[7] Smits, S.A., Leach, J., Sonnenburg, E.D., Gonzalez, C.G., Lichtman, J.S., Reid, G., Knight, R., Manjurano, A., Changalucha, J., Elias, J.E. and Dominguez-Bello, M.G., 2017. Seasonal cycling in the gut microbiome of the Hadza hunter-gatherers of Tanzania. Science, 357(6353), pp.802-806.

[8] Wan, C., Zhu, C., Jin, G., Zhu, M., Hua, J. and He, Y., 2021. Analysis of gut microbiota in patients with coronary artery disease and hypertension. Evidence-Based Complementary and Alternative Medicine, 2021(1), p.7195082.

[9] Yan, Q., Gu, Y., Li, X., Yang, W., Jia, L., Chen, C., Han, X., Huang, Y., Zhao, L., Li, P. and Fang, Z., 2017. Alterations of the gut microbiome in hypertension. Frontiers in cellular and infection microbiology, 7, p.381. 10.Yao, H., Wan, J.Y., Wang, C.Z., Li, L., Wang, J., Li, Y., Huang, W.H., Zeng,

[10] J., Wang, Q. and Yuan, C.S., 2018. Bibliometric analysis of research on the role of intestinal microbiota in obesity. PeerJ, 6, p.e5091.

[11] Jie, Z., Xia, H., Zhong, S.L., Feng, Q., Li, S., Liang, S., Zhong, H., Liu, Z., Gao, Y., Zhao, H. and Zhang, D., 2017. The gut microbiome in atherosclerotic cardiovascular disease. Nature communications, 8(1), p.845.

[12] Li, J., Zhao, F., Wang, Y., Chen, J., Tao, J., Tian, G., Wu, S., Liu, W., Cui, Q., Geng, B. and Zhang, W., 2017. Gut microbiota dysbiosis contributes to the development of hypertension. Microbiome, 5, pp.1-19.

[13] Chaplin, D.D., 2010. Overview of the immune response. Journal of allergy and clinical immunology, 125(2), pp.S3-S23.

[14] Stecher, B. and Hardt, W.D., 2008. The role of microbiota in infectious disease. Trends in microbiology, 16(3), pp.107-114.

[15] Macfarlane, S., Steed, H. and Macfarlane, G.T., 2009. Intestinal bacteria and inflammatory bowel disease. Critical reviews in clinical laboratory sciences, 46(1), pp.25-54.

[16] Mathisen, G.E. and Johnson, J.P., 1997. Brain abscess. Clinical Infectious Diseases, pp.763-779.

[17] Selhub, E., 2015. Nutritional psychiatry: Your brain on food. Harvard Health Blog, 16(11), p.2015.

[18] Cahill, T.J., Baddour, L.M., Habib, G., Hoen, B., Salaun, E., Pettersson, G.B., Schäfers, H.J. and Prendergast, B.D., 2017. Challenges in infective endocarditis. Journal of the american college of cardiology, 69(3), pp.325-344.

[19] Binder, C.J., Hörkkö, S., Dewan, A., Chang, M.K., Kieu, E.P., Goodyear, C.S., Shaw, P.X., Palinski, W., Witztum, J.L. and Silverman, G.J., 2003. Pneumococcal vaccination decreases atherosclerotic lesion formation: molecular mimicry between Streptococcus pneumoniae and oxidized LDL. Nature medicine, 9(6), pp.736-743.

[20] Stevens, D.L., 2003. Group A streptococcal sepsis. Current infectious disease reports, 5, pp.379-386.

[21] Kazankov, K., Jørgensen, S.M.D., Thomsen, K.L., Møller, H.J., Vilstrup, H., George, J., Schuppan, D. and Grønbæk, H., 2019. The role of macrophages in nonalcoholic fatty liver disease and nonalcoholic steatohepatitis. Nature reviews Gastroenterology & hepatology, 16(3), pp.145-159.

[22] Parsonnet, J., 1995. Bacterial infection as a cause of cancer. Environmental health perspectives, 103(suppl 8), pp.263-268.

[23] Shukla, R., Shukla, P., Behari, A., Khetan, D., Chaudhary, R.K., Tsuchiya, Y., Ikoma, T., Asai, T., Nakamura, K. and Kapoor, V.K., 2021. Roles of Salmonella typhi and Salmonella paratyphi in gallbladder cancer development. Asian Pacific Journal of Cancer Prevention: APJCP, 22(2), p.509.

[24] Labarca, G., Drake, L., Horta, G., Jantz, M.A., Mehta, H.J., Fernandez-Bussy, S., Folch, E., Majid, A. and Picco, M., 2019. Association between inflammatory bowel disease and chronic obstructive pulmonary disease: a systematic review and meta-analysis. BMC Pulmonary Medicine, 19, pp.1-9.

[25] Zhou, Y., Hong, Y. and Huang, H., 2016. Triptolide attenuates inflammatory response in membranous glomerulo-nephritis rat via downregulation of NF-κB signaling pathway. Kidney and Blood Pressure Research, 41(6), pp.901-910.

[26] Wang, J., Li, F., Wei, H., Lian, Z.X., Sun, R. and Tian, Z., 2014. Respiratory influenza virus infection induces intestinal immune injury via microbiota-mediated Th17 cell– dependent inflammation. Journal of Experimental Medicine, 211(12), pp.2397-2410 27.Wang, Q., Li, F., Liang, B., Liang, Y., Chen, S., Mo, X., Ju, Y., Zhao, H., Jia, H., Spector,

[27] T.D. and Xie, H., 2018. A metagenome-wide association study of gut microbiota in asthma in UK adults. BMC microbiology, 18, pp.1-7.