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THE ROLE OF MICROBIOLOGICAL FACTORS IN THE DEVELOPMENT AND COMPLICATIONS OF LIVER AND VASCULAR DISEASES OF THE HEART AND THE RELEVANCE OF MICROFLORA STABILIZATION

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Abstract. Human health depends on the gut microbiota for a variety of reasons, including nutritional digestion, intestinal epithelial barrier formation, immune function regulation, vitamin and hormone production, and the production of metabolites that interact with the host. The occurrence, development, and management of CVDs are strongly correlated with the gut microbiota, according to mounting data. In patients with CVDs and corresponding risk factors, the composition and ratio of gut microbiota have significant differences compared with their healthy counterparts. Therefore, some of the mechanisms regarding the occurrence and development of CVDs may be explained by gut microbiota dysbiosis, metabolites produced by the gut microbiota, and the associated signaling pathway. Numerous studies have also shown that the gut microbiota, the metabolites it produces, and associated signaling pathways are linked to both traditional and modern medicinal treatments for CVDs. Given that information, we summarized the latest advances in the current research regarding the effect of gut microbiota on health, the main cardiovascular risk factors, and CVDs, highlighted the roles and mechanisms of several metabolites, and introduced corresponding promising treatments for CVDs regarding the gut microbiota. Therefore, this review mainly focuses on exploring the role of gut microbiota related metabolites and their therapeutic potential in CVDs, which may eventually provide better solutions in the development of therapeutic treatment as well as the prevention of CVDs.

Keywords. Risk factors, cardiovascular diseases, metabolites, gut microbiota, dysbiosis, health.

Introduction. Heart failure (HF), hypertension, myocardial infarction (MI), and atherosclerosis are among the cardiovascular diseases (CVDs) that are increasingly associated with dysbiosis of the gut microbiota and its metabolic byproducts. Over 64 million people worldwide suffer from heart failure (HF), which is exacerbated by gut barrier disruption and systemic inflammation. In a similar vein, pro-inflammatory bacteria and decreased microbial diversity are associated with hypertension and MI, which raise the risk of cardiovascular disease and vascular inflammation. Gut dysbiosis also affects atherosclerosis, and important microbial metabolites such short-chain fatty acids (SCFAs) and trimethylamine-N-oxide (TMAO) are important in the pathophysiology of the disease [1,2,3]. New research shows that natural substances that alter the gut microbiota and provide cardioprotective benefits, such as flavonoids, omega-3 fatty acids, resveratrol, curcumin, and marine-derived bioactives, have therapeutic promise. Targeting dysbiosis may provide new preventive and therapeutic approaches, as our findings highlight the gut microbiota as a crucial regulator of cardiovascular health. To clarify underlying mechanisms and enhance microbiome-based therapies for better cardiovascular outcomes, more study is required. The gut's anaerobic and nutrient-rich environment is colonized by trillions of bacteria to create a robust intestinal physiological ecosystem. These groups are referred to as "gut microbiota," and "gut microbiomes" are the collective term for all of the genomes of microorganisms in the gut, including their DNA sequences and other genetic data.

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Bacteria with complex structures make up the majority of intestinal microbial communities in the human digestive tract [4,5,6,]. There are more than 1,000 species of intestinal bacteria, and the number reaches approximately 1014. The ratio of bacterial number to human cell number ranged from 10:1 to 1:1. It also contains 100-fold more genes than our own genome, which is called the second genome of the human body. In terms of the types of intestinal bacteria, the gut microbiota is mainly composed of Firmicutes, Bacteroidetes, Proteobacteria and Actinobacteria, and this composition is relatively stable in healthy individuals but not in patients with CVDs or other diseases. Among those gut microbiota, Firmicutes and Bacteroidetes in the large intestine account for about 90% of the total number of gut microbiota, and the ratio is a vital health indicator reflecting the condition of health and is also associated with the incidence of CVDs [7,8,9]. In order to create a dynamic and well-balanced microbial system, the gut microbiota also maintains an antagonistic or symbiotic relationship with its host. To date, establishing a clear and direct relationship between gut microbiota and corresponding diseases may be challenging due to the mixture of other non-host genes from viruses, fungi, and archaea. Thankfully, harmful non-host genes have been identified using blood or fecal samples and sophisticated sequencing technologies like 16S rRNA and metagenome. More importantly, the diversity and metabolites of normal gut microbiota are closely related to human health, and the imbalanced gut microbiota plays an important role in the occurrence and development of human diseases, in which the impact of gut microbiota on CVDs receives increasing attention [10,11,12]. Recent studies have highlighted the involvement of the microbiome in the pathophysiology of chronic diseases, with dysbiosis being associated with cancer, inflammatory bowel disease (IBD), obesity, and type 2 diabetes. Probiotics, prebiotics, and fecal microbiota transplantation are examples of novel therapies that may be discovered by comprehending microbiome-mediated pathways. The focus of this review is on CVDs and natural products with a microbiome focus as supplements to conventional therapies. Given the significant prevalence of CVDs worldwide and their links to inflammation and metabolism, it's critical to look into microbiome-based medicines as potential novel treatments. In order to investigate the function of gut microbiota in physical circumstances, pathological dysbiosis, and metabolites involved in the occurrence, development, and therapy of CVDs, we compiled the most recent research in this review [13,14,15]. In order to obtain a thorough grasp of the pathophysiology and mechanism of CVDs, we first thoroughly examined the impact of the gut microbiota on health, including its primary function and material metabolism. Next, we went into great detail about the impact of the major metabolites that the gut microbiota produces on a number of prevalent cardiovascular risk factors and the primary CVDs. In the end, we also examined and clarified the data about promising strategies for the prevention and treatment of CVDs, including food interventions. More significantly, we discussed how focusing on gut microbiota and its metabolites will be a fresh approach to CVD prevention and treatment [16,17,18].

The main purpose of this brief review is to analyze the role of microbiological factors in the development and complications of liver and vascular diseases of the heart and the relevance of microflora stabilization based on reputable scientific papers.

Contributions of Human Microbes. A varied microbial community vital to human health, the gut microbiota is associated with enhanced immunological response, better metabolism, and a lower risk of chronic illness. About 90% of the microorganisms in a healthy adult are composed primarily of *Bacillota* and *Bacteroidota*. More than 1000 species make up the microbiota, which is estimated to have 150 times as many genes as the human genome. Less than 10% of two people's microbiomes are alike, making each individual unique. *Lactobacillus, Clostridium, Ruminococcus* and *Faecalibacterium* are among the Bacillota that are responsible

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for the fermentation of fiber and the generation of SCFA, which in turn stimulates colonocyte energy and immunological regulation. Obesity is associated with elevated levels of *Bacillota*, which are also involved in energy metabolism [1,2,7,11]. The generation of SCFA, the integrity of the gut barrier, and the metabolism of carbohydrates are all impacted by *Bacteroidota*, which includes *Bacteroides* and *Prevotella*. Since its disturbance has been linked to metabolic illness, a balanced ratio of Bacillota to *Bacteroidota* is essential. Probiotic actinobacteria, especially *Bifidobacterium*, inhibit pathogens and modulate the immune system. Although protobacteria, such as *Escherichia, Enterobacter* and *Helicobacter*, are naturally found in small quantities, their overabundance has been linked to intestinal inflammation and illnesses like IBD and colorectal cancer. *Probiotics (Lactobacillus* and *Bifidobacterium*) are introduced by fermented foods including yogurt, kefir, kimchi, and sauerkraut, and they improve gut health and microbial diversity [19,20,21]. In addition to nutrition, a number of environmental, genetic, and lifestyle factors affect gut microbial populations. Protective bacteria like *Bifidobacteria* decrease with age, but pathogenic species that cause inflammation and decreased gut resilience increase. While a sedentary lifestyle encourages gut dysbiosis, exercise increases microbial diversity and SCFA output. By increasing intestinal permeability and pro-inflammatory bacteria, smoking and alcohol misuse have a detrimental effect on microbiota. Chronic microbial dysbiosis due to agents like antibiotics, PPIs, and NSAIDs increases vulnerability to infections like Clostridium difficile; genetic predisposition controls microbial colonization, which impacts immune function and metabolic competence; geographic locations, cultural practices, and environmental pollutants (e.g., heavy metals, pesticides, and microplastics) also control the composition of the microbiome, potentially causing inflammation and metabolic disease; and early-life exposures, such as breastfeeding, birth mode, and early-life antibiotic exposure, have a significant effect on microbiota diversity, affecting immune development and disease susceptibility [22,23,24].

The gut microbiota's function in both health and illness. According to studies, the fetus was exposed to bacteria before to birth, and during pregnancy, the fetus became colonized by anaerobic bacteria. According to conventional wisdom, the uterus is a sterile environment, and breastfeeding and other dietary feedings cause the intestinal microbiota to progressively colonize once the fetus is born. Over time, the gut microbiota's diversity and abundance grew during the newborn era, and during the early years, the diversity steadily developed and stabilized. As a result, in healthy people, these microorganisms compete and limit one another to preserve a typical dynamic equilibrium state. Commensals are another name for the bacteria that inhabit the human gut. Some beneficial commensal microorganisms, like *Roseburia intestinalis, Faecalibacterium prausnitzii* and *Akkermansia muciniphila (A. muciniphila),* may have anti-inflammatory properties and restore the normal function of the intestinal barrier [25,26,27]. Approximately 20% of the gut microbiota is composed of these commensal bacteria. By synthesising a range of vitamins, aiding in food digestion, generating lactic acid, encouraging intestinal peristalsis, preventing the growth of pathogenic microbiota, and stimulating the immune system, they are essential for preserving the physiological function of adult tissues and organs. Pathobionts also include other commensal bacteria as *Helicobacter pylori, Lactobacillus, Streptococcus, Escherichia coli,* segmented filamentous bacteria, and enterotoxigenic B. fragilis. They interact with the host to control the immune response and comprise over 70% of the entire gut microbiota. Physically, they are innocuous, but with environmental changes, they may have a pathogenic effect on the host. However, these pathobionts can quickly multiply significantly in cases of immunological failure and other clinical circumstances, or they might go from the intestine to other parts of the body and cause harm. In order to investigate the role of gut microbiota in health, we provide here the primary function of gut microbiota interacting with the

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host via controlling the intestinal mucosa barrier, immunological homeostasis, and nutritional metabolism (glucose, lipids, and protein). Next, we discuss how the gut microbiota contributes to the development of diseases, particularly cardiovascular disorders [28,29,30,31].

Gut microbiota with cardiovascular diseases (CVDs). With complex etiologies based on genetic, behavioral, and environmental factors, CVDs are a major source of morbidity and mortality globally. The idea that the wide variety of bacteria in the gastrointestinal system, known as the gut microbiota, significantly influences cardiovascular health is being more and more supported by data. The relationship between microbial composition and host metabolism has gained increasing attention, especially in connection with dysbiosis and its role in the pathophysiology of CVD. Through complex processes including inflammation, metabolism, and microbial dysbiosis, the gut microbiota plays a crucial role in the pathophysiology of CVDs like atherosclerosis, hypertension, myocardial infarction, and heart failure (HF). The clinical illness known as heart failure (HF) is characterized by decreased cardiac function, which lowers blood flow. Globally, there were more than 64 million instances in 2017; heart failure is still a serious health concern, and as people age and live longer, its prevalence is predicted to increase [32,33,34]. It has a significant financial impact as well; by 2030, healthcare spending in the US is expected to reach $69.8 billion. There is mounting evidence that gut dysbiosis contributes to the development of heart failure by upsetting homeostasis, increasing intestinal permeability, and causing systemic inflammation. The thickening of the intestinal wall, edema, and barrier failure are caused by HF-related anatomical and functional changes in the gastrointestinal tract, including as systemic blood redistribution, decreased cardiac output, and neuro-humoral activation. Gut-derived lipopolysaccharide (LPS) is another important factor that contributes to the advancement of HF. Systemic inflammation, oxidative stress, and fibrosis are brought on by LPS translocation into the circulatory system. Toll-like receptor 4 (TLR4) receives signals from LPS that cause cytokine release, arrhythmias, and cardiac damage, whereas MMP-mediated remodeling and oxidative imbalance worsen contractility. By raising immune cell infiltration, weakening the integrity of the gut barrier, and boosting inflammatory signals, gut dysbiosis also maintains systemic inflammation. Given the critical role inflammation plays in the development of HF, pharmacologically targeting inflammatory pathways may offer new treatment alternatives [17,21,28].

Changes in GM-mediated epigenetics in age-related heart failure. A decrease in the accuracy of epigenetic control, which results in abnormal gene expression patterns, is one of the many physiological changes associated with aging. The initiation and progression of age-related disorders, including heart failure, are intimately associated with epigenetic alterations, which are acknowledged as defining characteristics of aging. Nevertheless, little is known about the precise function of these alterations in heart aging. Although it is commonly known that epigenetic changes have a role in heart failure, it is still unknown which epigenetic modifications are the fundamental causes of the disease and which are merely secondary effects of aging. The epigenetic profiles of elderly people, who are most at risk of developing heart failure, differ from those of younger people. Chronic, long-term changes in gene expression may result from these age-related changes in epigenetic control. These changes not only mirror the heart's natural aging process but also increase preexisting susceptibilities to cardiac dysfunction [27,28,31]. However, utilizing human left ventricular biopsies or murine models, the majority of research on the epigenetic basis of HF has been done on either juvenile or adult patients. A crucial knowledge gap about how aging affects the epigenomic landscape in the setting of HF is highlighted by this emphasis on younger groups. Our capacity to completely understand the mechanisms underlying age-related heart failure is limited by the paucity of research on the epigenetic changes in the

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aging heart. Since our understanding of the mechanisms behind age-related heart failure is limited by our inability to adequately predict the epigenetic landscape in senior populations, it is imperative that these knowledge gaps be filled. Therefore, there aren't many direct investigations on how dysbiosis and GM-derived metabolites affect cardiac-epigenetic alterations, particularly in older people. Nonetheless, there is growing evidence that GM and epigenetic changes in systemic situations are related [1,2,14]. According to a different study, the GM and four epigenetic clocks are causally related and can be influenced by a number of inflammatory stimuli. Although the precise effect of these alterations on the cardiac epigenome is still unknown, these results imply that GM may have an impact on age-related epigenetic regulation. It is conceivable that the impacts shown in systemic contexts, such as blood-based epigenetic clocks, may also apply to the heart given the lack of studies specifically examining the interaction between the GM and the cardiac epigenome in the aged. Without concrete proof, it is still unclear how the dysbiosis frequently seen in the elderly might affect the epigenetic modifications that predispose people to heart failure. To determine whether GM affects the heart's epigenetic control as we age and how this relationship may contribute to the onset and progression of heart failure in older populations, more research that fills this knowledge gap is required [23,29, 34,35].

Discussion. The host's intestinal barrier, immune system, and material metabolism are all regulated by the mutualistic environment that the gut microbiota creates with the host. On the other hand, dysbiosis of the gut microbiota causes inflammation, interferes with normal function, and generates toxic compounds that lead to disease. The connection between gut microbiota and illnesses is intriguing because it raises the possibility that the gut microbiota could be a new therapeutic target. In order to stop the progression of diseases, it would be very beneficial to determine the underlying molecular relationship between gut microbiota and illnesses. Food and medications are two of the many external factors that have an impact on the human gut flora. In addition to preserving a normal energy balance, a nutritious diet can control the diversity and makeup of the human gut microbiota, which in turn can influence the onset and progression of disease [11,14,18]. For CVDs, for instance, the Mediterranean diet offers several advantages, including reducing inflammatory markers and blood cholesterol. A calorie-restricted diet, a higher dietary fiber content, and exercise training can also alter energy balance, improving health and lowering the risk of disease. In addition, sex is a major factor in this situation. The varying rates of metabolic disorders seen in males and females, as well as the sex-specific impact of diet on GM composition, have been shown to be related to GM in previous studies. In fact, because of differences in host metabolism and gut composition, men and women create different microbiota-derived metabolites. For instance, SCFAs levels, such as butyrate and propionate, may differ and influence sex-specific inflammation responses [21,25,27]. These microbial metabolites can modulate hormone pathways differently by sex, impacting conditions like obesity, cardiovascular and mental health, and they also can influence DNA methylation and histone modification, possibly affecting longevity and disease susceptibility differently in men and women. These interactions imply that age- and gender-specific health challenges may benefit from both age- and personalized approaches to health interventions; however, more research is needed to determine how sex relates to GM's role in HF onset and progression, which is mediated by epigenetic modifications. In order to prevent and treat heart failure (HF) and improve patient outcomes and prognosis, particularly for the elderly, who bear the greatest burden of the disease, it is imperative that these gaps be filled. We can customize therapy for each patient according to their distinct microbial profiles and epigenetic signatures by detecting particular metabolite-induced epigenetic alterations. The intricacies of this interaction require a multidisciplinary strategy that integrates knowledge from cardiology, genetics, and microbiology

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[13,14,19]. For a number of reasons, it is imperative to look into the epigenetic impact of GM-derived metabolites on HF, particularly in the older population. The pathophysiology of age-related heart disease can be better understood, novel biomarkers and treatment targets can be found, and eventually, the prognosis of older persons with HF can be improved. Furthermore, the investigation of epigenetic pathways may yield novel biomarkers for the prognosis and early diagnosis of heart failure. The development of medications that can alter gene expression and enhance heart function may also find novel therapeutic targets in these systems [30,31,32].

Conclusion. There is mounting evidence of the complex connection between dysbiosis of the gut microbiota and cardiovascular diseases (CVDs), such as atherosclerosis, heart failure, hypertension, and MI. Through metabolites produced by microbes, such as TMAO and SCFAs, dysbiosis promotes vascular dysfunction, systemic inflammation, and the advancement of disease. A promising therapeutic approach is presented by the potential of natural substances such as flavonoids, omega-3 fatty acids, resveratrol, curcumin, and marine-derived bioactives to restore microbial balance and provide cardioprotective benefits. Although focusing on the gut microbiota presents new approaches to managing and preventing CVD, further study is required to clarify the underlying mechanisms and create therapies based on the microbiome.

In order to create more effective targeted disease intervention programs and generate fresh concepts for enhancing health and treating CVDs, high-tech methods involving bacterial gene sequencing should be employed in the future to identify the composition and role of gut microbiota metabolites. The emergence of a new generation of metagenomics and the development of bioinformatics will help us to further study the production and mechanism of microbial metabolites. To further confirm how the gut microbiota transforms endogenous and dietary molecules into metabolites that communicate with the host's peripheral organs and tissues, numerous high-quality studies are still required. This will help develop therapies that target the gut microbiota and identify novel approaches to the prevention and treatment of CVDs.

In conclusion, it is critical to look into the epigenetic effects of GM-derived metabolites on heart failure, particularly in the elderly, for a number of reasons. It has the potential to improve the prognosis of elderly patients with heart failure, uncover novel biomarkers and treatment targets, and deepen our understanding of the pathophysiology of age-related heart disease. Furthermore, novel biomarkers for the prognosis and early diagnosis of HF may be discovered as a result of research into epigenetic pathways. Furthermore, these processes can serve as novel therapeutic targets for the creation of medications that might alter gene expression and enhance heart function.

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