Biomedical Journal of Indonesia
Journal Homepage: https://bji-fk.ejournal.unsri.ac.id

EDITORIAL

Potential Modulation of Gut Microbiota by Phytochemicals in Managing Noncommunicable Diseases

Rizki Andini Nawawi[1*]

[1]Department of Clinical Microbiology, Faculty of Medicine, Universitas Sriwijaya, Palembang, Indonesia

ARTICLE INFO

Keywords:
Gut microbiota
Hypertension
Type 2 diabetes
Phytochemicals

Corresponding author:
Rizki Andini Nawawi

E-mail address:
rizkiandininawawi@fk.unsri.ac.id

https://doi.org/10.32539/BJI.v11i3.250

Received 3 March 2025;
Accepted 26 June 2025

ABSTRACT

Noncommunicable diseases, such as hypertension and type 2 diabetes, remain a leading cause of death worldwide, especially in low- and middle-income countries. Recent research trends revealed possible involvement of the gut microbiota in the pathogenesis of these noncommunicable diseases. This article provides a review of recent research trends on the human gut microbiota's involvement in hypertension and type 2 diabetes, and the potential of its modulation by natural products. Several mechanisms are proposed to explain how the gut microbiota contributes to the development of hypertension and type 2 diabetes, one of which is by changes in short-chain fatty acids (SCFAs) production. Phytochemicals targeting modulation of the gut microbiota and SCFAs production may show potential in prevention and therapy of hypertension and diabetes. This may be achieved by affecting the composition of the gut microbiota itself and by modulating the microbiota's metabolic pathways, such as by stimulating synthesis of SCFAs. However, future studies are still needed considering the complexity of factors affecting gut microbiota, as well as the need to develop reproducible methods in studying the gut microbiota and its metabolites in order to further elucidate the role of gut microbiota in health and disease, in order to achieve optimal benefits and better clinical outcomes.

1. Introduction

Noncommunicable diseases remain a leading cause of death globally. Noncommunicable diseases caused more than 43 million deaths in 2021, where nearly three quarters of these deaths happen in low- and middle-income countries.[1] In Indonesia, there has been a shift from infectious diseases to noncommunicable diseases as main cause of mortality over the past 3 decades. Two diseases of significant interest are hypertension and type 2 diabetes, as they represent a high burden of disease and may lead to cardiovascular complications and deaths.[1,2] In addition, hypertension and diabetes, along with smoking and ambient particulate matter air pollution, represent the leading risk factors driving the global burden of disease. These risk factors affect each level of the Socio-demographic Index.[3]

Humans live alongside microorganisms, encompassing bacteria, protozoa, archaea, other eukaryotes, and viruses. These microorganisms form a thriving microecosystem known as microbiota, and reside in various sites of the human body. One of the sites harboring an important microbiota population is the gastrointestinal tract. The symbiotic relationship of microbiota and the human host are reflected in various complex physiological processes. Imbalances in the microbiota, or dysbiosis, have been linked to the pathogenesis of human diseases, including important noncommunicable diseases.[4] Gut microbiota may influence human health and sickness through an interplay with various environmental and host factors, even mediating effects of medicines.[5] The effects are exerted through signaling mechanisms between the gut microbiota and host cells, mediated by various messenger molecules and metabolites. In particular, short-chain fatty acids (SCFAs) such as acetate, propionate, and butyrate, are thought to play important roles in the physiological processes and pathophysiology of diseases affecting the human host, both locally and systemically.[6]

Diet is known to influence the composition and

physiology of gut microbiota. Some naturally occurring compounds widely present in the diet, such as phytochemicals, may have a beneficial effect in reducing risks of chronic illnesses. One of the mechanisms through which phytochemicals and natural compounds from the diet exert their actions is by creating favorable conditions for select bacterial populations, regulating metabolites, or restoring the diversity of the gut microbiota.[5,7] Phytochemicals are relatively poorly absorbed owing to their structural complexity. This may lead to longer retention times in the intestines, allowing the phytochemicals extracted from food intake to interact with and influence the microbiota.[5,8] This article aims to review recent research trends on the human gut microbiota's involvement in hypertension and diabetes mellitus, and to explore the potential of its modulation by natural products.

2. The Role of Gut Microbiota in Noncommunicable Diseases

Gut Microbiota in Hypertension

Dysbiosis of the gut microbiota had been observed in both animal models and human subjects with hypertension. Several notable changes in the gut microbiota included decreased diversity, compositional changes of several taxa, changes in microbial structure and function, and shifts in microbial interactions.[9] A study on hypertensive mice reported a drastic decrease in microbial richness and diversity of gut microbiota in comparison to healthy controls. *Prevotella* and *Klebsiella* overgrowth, along with decrease in bacteria associated with healthy gut microbiota and disease-linked changes in microbial functions, were observed in both pre-hypertensive and hypertensive models.[10] A meta-analysis from 8 human studies also discovered a significant change in gut microbiota richness and diversity in hypertensive patients. Some bacteria, such as *Catabacter, Robinsoleilla, Serratia, Enterobacteriaceae, Ruminococcus torques, Parasutterella, Escherichia, Shigella,* and *Klebsiella* showed increased abundance. In contrast, decreased abundance of *Sporobacter, Roseburia hominis, Romboutsia* spp., and other *Roseburia* species were also observed. These alterations varied by several factors, such as diet, age, ethnicity, and hypertension severity.[11]

There are several possible mechanisms for the gut microbiota's involvement in hypertension pathogenesis, such as SCFAs-mediated signaling, brain-gut-bone marrow interaction, and inflammation.[12] SCFAs, such as acetate, butyrate, and propionate, are products of dietary fiber fermentations. SCFAs act by two primary receptors, the olfactory receptor 78 (Olfr78) and G-protein-coupled receptor 41 (Gpr41). These two receptors contribute to different effects on blood pressure. The binding of SCFAs to Olfr78 elevates blood pressure, while Gpr41 lowers baseline blood pressure. So, balanced expression and activation of these two receptors are necessary to maintain physiological blood pressure. However, even in balanced expression of Olfr78 and Gpr41, an imbalance in SCFAs due to changes in the gut microbiota, poor absorption and high excretion of SCFAs, may affect blood pressure.[9,12] A human study on 441 participants reported that SCFAs concentrations were inversely associated with microbiota diversity. In addition, each SCFA was found to be associated with unique microbial taxa. Higher fecal SCFA concentrations were also associated with hypertension.[13] SCFAs also exert other protective effects on the cardiovascular system through their anti-inflammatory properties.[14]

In addition to SCFAs, other small molecule metabolites produced by the gut microbiota are also associated with the pathogenesis of hypertension.[5] Tri-methylamine-N-oxide (TMAO), a metabolite of trimethylamine (TMA) produced by gut microbiota, might contribute to the pathogenesis of hypertension by prolonging the hypertensive effect of angiotensin II. Elevated TMAO might also act on blood pressure by stimulating vasopressin release and upregulating aquaporin-2 expression in the apical membrane of chief cells of the renal collecting duct, therefore increasing sodium-water retention.[5,15] In addition, TMAO enhances foam cell formation, reduces reverse cholesterol transport, and induces platelet aggregation, therefore exerting atherogenic and thrombogenic effects.[16] The possible role of bile acids and H_2S in hypertension pathogenesis might be through impairment in vasomotor regulation.[5] Indoxyl sulfate and p-cresyl sulfate are also known as uremic toxins, which may be produced by gut microbiota metabolism. They possess proatherosclerotic, prothrombotic and proinflammatory properties, which are involved in arterial calcification. They can also contribute to endothelial dysfunction by triggering oxidative stress.[15] In addition, a number of commensal bacterial genera are able to synthesize vasoactive hormones, such as *Escherichia, Lactobacillus, Bifidobacterium and Streptococcus.* The vasoactive hormones, such as serotonin, dopamine and norepinephrine, may more directly exert their effect on blood pressure by vasoconstrictive mechanisms.[15]

Gut Microbiota in Type 2 Diabetes Mellitus

Gut microbiota dysbiosis in type 2 diabetes mellitus had also been observed. Most abundant constituents of the gut microbiota are classified under 2 phyla, Firmicutes and Bacteroidetes. The ratio of Firmicutes to Bacteroidetes was shown to be increased in diabetics in comparison to healthy people.[8] A systematic review from 42 human studies reported that *Bifidobacterium, Bacteroides,*

Faecalibacterium, Akkermansia and Roseburia were negatively associated with type 2 diabetes. In contrast, *Ruminococcus, Fusobacterium* and *Blautia* had positive association with type 2 diabetes.[17] In addition, increased abundance of *Proteobacteria, Clostridiales* and the species *Lactobacillus gasseri* and *Streptococcus mutans* were also reported in type 2 diabetics.[18]

The gut microbiota may contribute to the development of type 2 diabetes through several mechanisms, such as by modulating inflammation, interactions with dietary constituents, and exerting its effects on energy homeostasis in the human host through gut permeability, metabolism of glucose and lipid, and insulin sensitivity.[17] Expression of oxidative stress-related microbial genes is increased in several pathological mechanisms leading to type 2 diabetes, such as insulin resistance, β cell dysfunction, and impaired glucose tolerance.[18]

SCFAs are also thought to play an important role in the pathogenesis of type 2 diabetes. A particular SCFA of interest is butyrate, which supports proper function of β-cells in the pancreas, especially after food intake. Butyrate affects inflammation by modulating intestinal macrophage functions and downregulating pro-inflammatory mediators induced by LPS. It also activates intestinal gluconeogenesis and, as a result, ameliorates glucose homeostasis. Changes in the gut microbiota composition affecting butyrate-producing species had been consistently reported in studies on type 2 diabetics.[19,20]

A large-scale study involving the LifeLines-DEEP (LL-DEEP) cohort investigated the association between SCFAs, specifically butyrate and propionate, and type 2 diabetes risk. The study reported an association between increased gut butyrate production and improved insulin response after an oral glucose test, while disturbances in production or absorption of propionate indicated an increased type 2 diabetes risk. In addition, genetically-influenced changes favoring production of butyrate in the gut microbiome appeared to show beneficial effects on β cell function, while host genetic variation affecting production of propionate (by increasing propionate production or impairing its absorption) impacted risk of type 2 diabetes.[20]

In addition to SCFAs, several metabolites of gut microbiota, including TMAO, LPS, uremic toxins derived from aromatic amino acids, and related metabolites are also thought to contribute to the development of type 2 diabetes. Release of LPS from gut microbiota into the blood circulation leads to inflammatory response through formation of activated (TLR4-MD-2-LPS)2 complex and downstream NF-kB activation. The initial inflammatory response may disrupt the intestinal barrier, allowing access to more microbial components in the intestinal lumen into the host system and triggering systemic inflammation.[21,22] TMAO plays an important role in glucose metabolism and target organs dysfunction. Uremic toxins, such as indoxyl sulfate and p-cresyl sulfate, are known to exert multi-toxicities in glomerular cells, possibly contributing to renal complications of type 2 diabetes as well.[21]

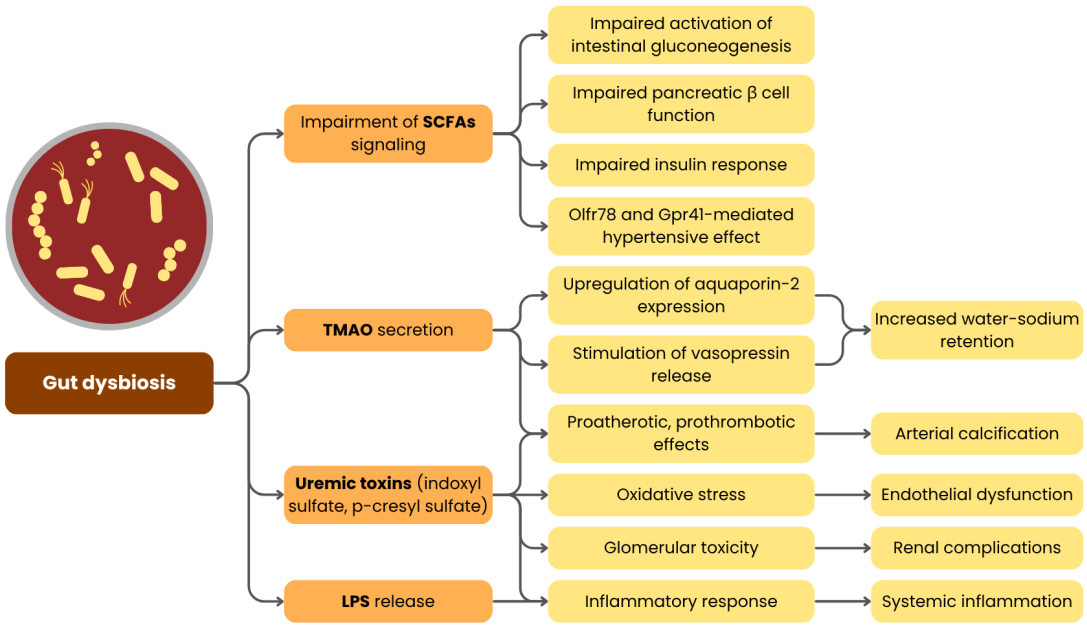

Figure 1. The effects of gut dysbiosis affecting pathogenesis of hypertension and type 2 diabetes mellitus
SCFAs: short-chain fatty acids, TMAO: Tri-methylamine-N-oxide, LPS: lipopolysaccharide

3. Modulation of Gut Microbiota by Phytochemicals in Noncommunicable Diseases

Microbiota Composition Shifting

Several phytochemicals obtained from various medicinal plants and foods have been investigated for their beneficial modulation of the gut microbiota in noncommunicable diseases. For instance, phytochemicals obtained from garlic and its products showed modulatory activities on the gut microbiota in hypertension through several mechanisms. Garlic supplementation in the diet was reported to increase the abundance of *Lachnospiraceae*, which is a member of the gut microbiota capable of synthesizing SCFAs. In addition, garlic was shown to inhibit growth of select microorganisms in the gut, allowing more growth of *Lactobacillus* bacteria. The genus *Lactobacillus* consist of lactic acid bacteria with an essential role in maintaining gastrointestinal barrier integrity.[23] In addition, *Lactobacillus* bacteria are able to affect blood pressure via renin-angiotensin-aldosterone system modulation. Some species of *Lactobacillus* synthesize short peptides such as Val-Pro-Pro and Ile-Pro-Pro, which lower blood pressure through inhibition of angiotensin-converting enzyme (ACE). Enhancement of mineral and phytoestrogens assimilation by *Lactobacillus* may also exert a vasodilatory effect.[14] Garlic additionally exerts other vasoactive effects. Raw garlic contains allicin, which is a volatile organosulfur compound. Allicin inhibits the synthesis of TMAO by the gut microbiota, which may inhibit TMAO's atherogenic and thrombogenic effects. Furthermore, aging garlic may convert the allicin into S-allylcystein, a phytochemical with known vasoactive properties.[23,24]

Tea has previously shown antihypertensive activity mainly mediated by polyphenols, especially epigallocatechin gallate (EGCG).[25] However, tea may also exert its antihypertensive effect through shifting the composition of certain genera in the gut microbiota. An in vivo study in male Wistar rats fed with high-salt diet also reported oolong tea and green tea exerted a significant preventive effect against hypertension following 8 weeks of intervention. The green tea group showed a significant enrichment of the *Enterococcus* genus, while the oolong tea group had specific and significant enrichment for *Allobaculum, Paraprevotella, Oscillospira, Bifidobacterium*, and *Ruminococcus*.[26]

Extracts obtained from goji berry (*Lycium barbarum*) leaf also showed modulatory activity on the gut microbiota and its composition. *Lycium barbarum* leaf extract contains several polyphenols, such as neochlorogenic acid, chlorogenic acid, caffeic acid, and rutin. After 4 weeks of treatment with *Lycium barbarum* leaf extract, the abundance of *Marvinbryantia, Parasutterella*, *Prevotellaceae, Blautia*, *Ruminococcus*, and *Coprococcus* were restored in type 2 diabetes rat models.[27]

Modulation of SCFA synthesis

Modulation of SCFA synthesis by phytochemicals may also be a promising strategy in managing hypertension and type 2 diabetes. Administration of *Curcuma longa L.* ethanol-acetic acid-water extract into GIS1 colon simulators containing fecal microbiota sampled from hypertensive volunteer patients showed a positive modulation of SCFAs production. Curcumin, a constituent of *Curcuma longa* L. extract, stimulated the formation of butyric and propionic acids. Metabolomics-wise, this effect was shown to be similar to that exerted by dietary fibers, and the positive effects were dose-dependent.[28]

Mogrosides with 1–3 glucosyl residues (L-SGgly) obtained from the Luo-Han-Guo fruits (*Siraitia grosvenorii*) extract also showed modulatory effects on SCFAs production. Administration of both the fruit extract and the L-SGgly on type 2 diabetes rats for 14 days significantly increased SCFAs concentration, chiefly acetate and butyrate, as detected in the rats' feces. In addition to SCFAs, L-SGgly also decreased deoxycholic acid and 1β-hydroxycholic acid levels.[29]

4. Limitations and Challenges

Studies on the involvement of gut microbiota on the pathogenesis of noncommunicable diseases, as well as the modulatory effects of phytochemicals on the gut microbiota, still face several limitations. For instance, many of the cited studies on how gut microbiota interacts with the host used animal models, especially murine models. This may limit our current ability to translate these findings to the human microbiota. The gut microbiota is a dynamic microecosystem affected by various factors. A plethora of host and environmental factors, such as age, diet, medications, exercise, and comorbidities may influence the composition and metabolic activities of the gut microbiota. In addition, host genetics, stress, and changes in the circadian rhythm may also affect the gut microbiota.[11,22] How drugs other than antibiotics interact with the gut microbiota may also affect the composition of gut microbiota. For example, the oral antidiabetic drug metformin has been reported to be linked with higher abundances of the mucin-degrading *Akkermansia muciniphila* and some SCFA-producing genera, such as *Megasphaera, Bifidobacterium* and *Prevotella.*[18] These factors are often not yet considered in animal studies or existing trials involving human participants. This presents a challenge for future studies, especially those involving human participants, to consider these factors.

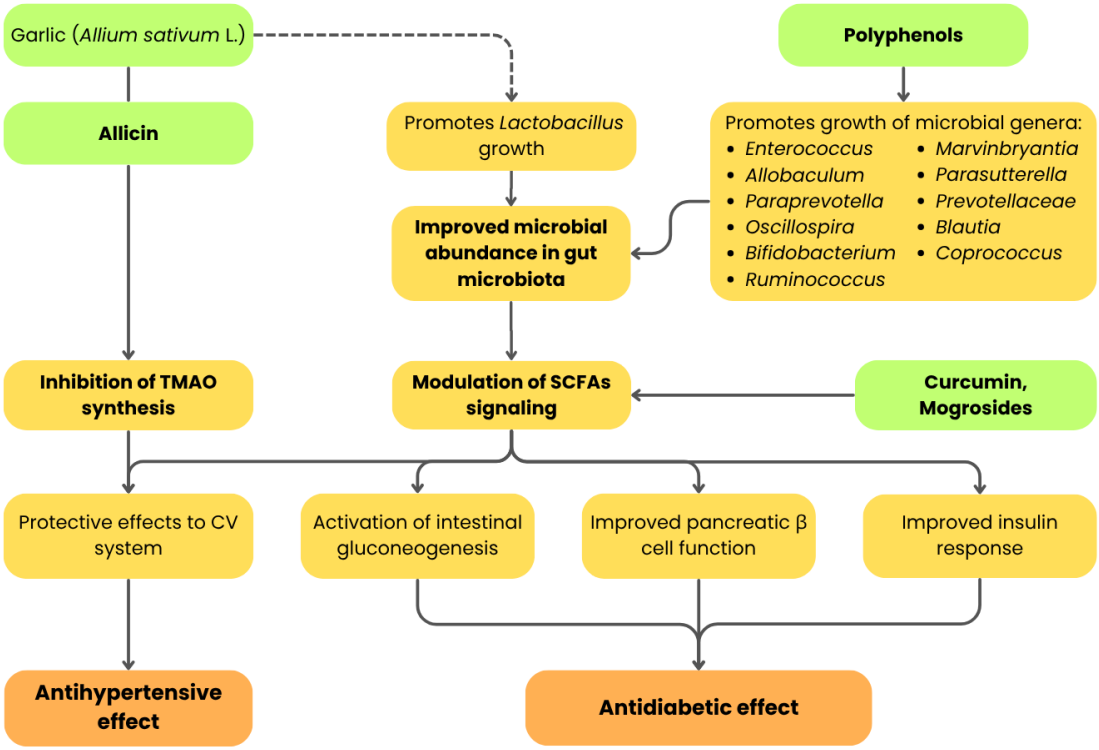

Figure 2. Modulation of gut microbiota and its products by phytochemicals. SCFAs: short-chain fatty acids TMAO: Tri-methylamine-N-oxide, CV: cardiovascular

Most studies use fecal sampling to determine the composition of the gut microbiota, or its metabolites. Mucosal biopsy may present a more ideal method to study microbiota composition and diversity, considering mucosal microbiota may differ from stool microbiota. However, mucosal sampling is not without its own limitations. Biopsy may cause the biofilm to be disrupted. Bowel preparation prior to colonoscopy and biopsy may also impact the sampled gut microbiota.[11] In addition, differences in microbiota abundance and diversity may also be affected by the study methods. For instance, studies have various, non-comparable DNA extraction protocols, sampling methods, and detection methods. These are further affected by varying sample sizes, interpersonal variation, or environmental factors.[19]

Phytochemicals may influence the gut microbiota and the host by a range of possible mechanisms, ranging from antioxidant activity, acting as substrates to directly influence microbial composition and abundance in a way similar to prebiotics, to inhibitory activity to pathogenic species by bactericidal or bacteriostatic means. These may also be influenced by synergism or additive effects from interacting with other dietary compounds or microbial metabolites.[30] In addition, like any other xenobiotics entering the human body, phytochemicals may also undergo metabolism by either the host or the microbiota. For instance, most polyphenols are degraded and absorbed in the colon after metabolism by local microbiota.[31]

Many studies on how plant products influence the gut microbiota are based on extracts or fractions, with the exact class or phytochemical compound acting on the gut microbiota and its mediated signaling axes remaining to be elucidated. Presently, the development of advanced technologies in metagenomics and metabolomics could aid in identifying and selecting biomarkers and possible targets in relation to dysbiosis and its effects on host metabolism. Together, these present a wide opportunity to identify potential phytochemical compounds from plant extracts, their pharmacokinetics on the host, and their molecular mechanisms of action to enable a more targeted therapeutic approach, both on the gut microbiota and host metabolism.

5. Conclusion

Phytochemicals targeting modulation of the gut microbiota may show potential in prevention and therapy of hypertension and type 2 diabetes. This may be achieved by affecting the composition of the gut microbiota, either by promoting growth and increasing abundance of select bacterial genera or species, or by suppressing pathogenic species. In addition, phytochemicals may act by modulating the microbiota's metabolic pathways, such as SCFAs synthesis.

However, in order to achieve optimal benefits

and better clinical outcomes, future studies are still needed considering the complexity of factors affecting gut microbiota, as well as the need to develop reproducible methods in studying the gut microbiota and its metabolites in order to further elucidate the role of gut microbiota in health and disease. In addition, identification of phytochemicals with good potential for gut microbiota modulation, as well as elucidation of their pharmacokinetics and molecular mechanisms of action, are also gaps that future studies need to address.

6. Author Contribution

R.A.N. conceived the idea and wrote the manuscript.

7. Conflict of Interest

None.

8. Acknowledgments

The author wishes to thank dr. Ridwan Balatif for his valuable insights in the writing of this manuscript.

9. References

1. World Health Organization. Noncommunicable diseases. World Health Organization. 2024. Accessed February 12, 2025. https://www.who.int/news-room/fact-sheets/detail/noncommunicable-diseases
2. BKPK Kementerian Kesehatan RI. Prevalensi, Dampak, serta Upaya Pengendalian Hipertensi dan Diabetes di Indonesia. In: Laporan Tematik Survei Kesehatan Indonesia Tahun 2023. Kementerian Kesehatan Republik Indonesia; 2024:67-88.
3. Institute for Health Metrics and Evaluation. Global Burden of Disease 2021: Findings from the GBD 2021 Study. IHME; 2024.
4. Qadri H, Shah AH, Almilaibary A, Mir MA. Microbiota, natural products, and human health: exploring interactions for therapeutic insights. Front Cell Infect Microbiol. 2024;14:1371312. doi:10.3389/fcimb.2024.1371312
5. Yang Z, Wang Q, Liu Y, et al. Gut microbiota and hypertension: association, mechanisms and treatment. Clin Exp Hypertens. 2023;45(1):2195135. doi:10.1080/10641963.2023.2195135
6. Overby HB, Ferguson JF. Gut Microbiota-Derived Short-Chain Fatty Acids Facilitate Microbiota:Host Cross talk and Modulate Obesity and Hypertension. Curr Hypertens Rep. 2021;23(2). doi:10.1007/s11906-020-01125-2
7. Santhiravel S, Bekhit AEDA, Mendis E, et al. The Impact of Plant Phytochemicals on the Gut Microbiota of Humans for a Balanced Life. Int J Mol Sci. 2022;23(15):8124. doi:10.3390/ijms23158124
8. Sharma BR, Jaiswal S, Ravindra PV. Modulation of gut microbiota by bioactive compounds for prevention and management of type 2 diabetes. Biomed Pharmacother. 2022;152:113148. doi:10.1016/j.biopha.2022.113148
9. Guo Y, Li X, Wang Z, Yu B. Gut Microbiota Dysbiosis in Human Hypertension: A Systematic Review of Observational Studies. Front Cardiovasc Med. 2021;8(May). doi:10.3389/fcvm.2021.650227
10. Li J, Zhao F, Wang Y, et al. Gut microbiota dysbiosis contributes to the development of hypertension. Microbiome. 2017;5(1):1-19. doi:10.1186/s40168-016-0222-x
11. Naik SS, Ramphall S, Rijal S, et al. Association of Gut Microbial Dysbiosis and Hypertension: A Systematic Review. Cureus. 2022;14(10):1-11. doi:10.7759/cureus.29927
12. Li J, Yang X, Zhou X, Cai J. The Role and Mechanism of Intestinal Flora in Blood Pressure Regulation and Hypertension Development. Antioxidants Redox Signal. 2021;34(10):811-830. doi:10.1089/ars.2020.8104
13. de la Cuesta-Zuluaga J, Mueller NT, Álvarez-Quintero R, et al. Higher fecal short-chain fatty acid levels are associated with gut microbiome dysbiosis, obesity, hypertension and cardiometabolic disease risk factors. Nutrients. 2019;11(1). doi:10.3390/nu11010051
14. Hamdi IA, Panggabean RI, Theresa CC, Nainggolan BWM, Balatif R. A Review of the Therapeutic Effects of Garlic in Lowering Blood Pressure: A Comprehensive Analysis of Recent Mechanisms and Existing Clinical Data. Biomed J Indones. 2025;11(1):26-37. doi:10.32539/bji.v11i1.211
15. Tokarek J, Budny E, Saar M, et al. Does the Composition of Gut Microbiota Affect Hypertension? Molecular Mechanisms Involved in Increasing Blood Pressure. Int J Mol Sci. 2023;24(2). doi:10.3390/ijms24021377
16. Panyod S, Wu WK, Chen PC, et al. Atherosclerosis amelioration by allicin in raw garlic through gut microbiota and trimethylamine-N-oxide modulation. npj Biofilms Microbiomes. 2022;8(1). doi:10.1038/s41522-022-00266-3
17. Gurung M, Li Z, You H, et al. Role of gut microbiota in type 2 diabetes pathophysiology. eBioMedicine. 2020;51:102590. doi:10.1016/j.ebiom.2019.11.051
18. Barlow GM, Celly S, Mathur R. Changes in the Gut Microbiome as Seen in Diabetes and Obesity. In: Pimentel M, Mathur R, Barlow GM, eds. Clinical Understanding of the Human Gut

Microbiome. Springer Nature Switzerland; 2023:61-81. doi:10.1007/978-3-031-46712-7_5
19. Bielka W, Przezak A, Pawlik A. The role of the gut microbiota in the pathogenesis of diabetes. Int J Mol Sci. 2022;23(1):1-20. doi:10.3390/ijms23010480
20. Sanna S, van Zuydam NR, Mahajan A, et al. Causal relationships among the gut microbiome, short-chain fatty acids and metabolic diseases. Nat Genet. 2019;51(4):600-605. doi:10.1038/s41588-019-0350-x
21. Lu J, Ma KL, Ruan XZ. Dysbiosis of Gut Microbiota Contributes to the Development of Diabetes Mellitus. Infect Microbes Dis. 2019;1(2):43-48. doi:10.1097/IM9.0000000000000011
22. Sharma S, Tripathi P. Gut microbiome and type 2 diabetes: where we are and where to go? J Nutr Biochem. 2019;63:101-108. doi:10.1016/j.jnutbio.2018.10.003
23. Lim WQ, Cheam JY, Law JWF, Letchumanan V, Lee LH, Tan LTH. Role of Garlic in Chronic Diseases: Focusing on Gut Microbiota Modulation. Prog Microbes Mol Biol. 2022;5(1):1-12. doi:10.36877/pmmb.a0000271
24. Ried K, Travica N, Sali A. The Effect of Kyolic Aged Garlic Extract on Gut Microbiota, Inflammation, and Cardiovascular Markers in Hypertensives: The GarGIC Trial. Front Nutr. 2018;5(December):1-14. doi:10.3389/fnut.2018.00122
25. Balatif R, Lubis NDA. Recent Literature Review: The Effects of Tea Consumption on Hypertension. Amerta Nutr. 2024;8(3):468-479. doi:10.20473/amnt.v8i3.2024.468-479
26. Ye X, Tang X, Li F, et al. Green and Oolong Tea Extracts With Different Phytochemical Compositions Prevent Hypertension and Modulate the Intestinal Flora in a High-Salt Diet Fed Wistar Rats. Front Nutr. 2022;9. doi:10.3389/fnut.2022.892801
27. Zhao X, Guo S, Lu Y, et al. *Lycium barbarum* L. leaves ameliorate type 2 diabetes in rats by modulating metabolic profiles and gut microbiota composition. Biomed Pharmacother. 2020;121:109559. doi:10.1016/j.biopha.2019.109559
28. Vamanu E, Gatea F, Sârbu I, Pelinescu D. An In Vitro Study of the Influence of *Curcuma longa* Extracts on the Microbiota Modulation Process, In Patients with Hypertension. Pharmaceutics. 2019;11(4):191. doi:10.3390/pharmaceutics11040191
29. Zhang Y, Peng Y, Zhao L, Zhou G, Li X. Regulating the gut microbiota and SCFAs in the faeces of T2DM rats should be one of antidiabetic mechanisms of mogrosides in the fruits of *Siraitia grosvenorii*. J Ethnopharmacol. 2021;274(March):114033. doi:10.1016/j.jep.2021.114033
30. Dingeo G, Brito A, Samouda H, Iddir M, La Frano MR, Bohn T. Phytochemicals as modifiers of gut microbial communities. Food Funct. 2020;11(10):8444-8471. doi:10.1039/d0fo01483d
31. Luo B, Wen Y, Ye F, et al. Bioactive phytochemicals and their potential roles in modulating gut microbiota. J Agric Food Res. 2023;12(November 2022):100583. doi:10.1016/j.jafr.2023.100583