ISSN 1948-9358 (online)

World Journal of *Diabetes*

World J Diabetes 2024 October 15; 15(10): 2002-2156

Published by Baishideng Publishing Group Inc

World Journal of Diabetes

Contents

Monthly Volume 15 Number 10 October 15, 2024

EDITORIAL

REVIEW

ORIGINAL ARTICLE

Retrospective Study

Observational Study

World Journal of Diabetes

Monthly Volume 15 Number 10 October 15, 2024

Contents

World Journal of Diabetes

Monthly Volume 15 Number 10 October 15, 2024

Contents

ABOUT COVER

Peer Review of *World Journal of Diabetes*, Tao-Hsin Tung, PhD, Researcher, Director, Epidemiologist, Evidence-based Medicine Center, Taizhou Hospital of Zhejiang Province Affiliated to Wenzhou Medical University, Taizhou 317000, Zhejiang Province, China. dongdx@enzemed.com .

AIMS AND SCOPE

The primary aim of *World Journal of Diabetes* (*WJD*, *World J Diabetes*) is to provide scholars and readers from various fields of diabetes with a platform to publish high-quality basic and clinical research articles and communicate their research findings online.

WJD mainly publishes articles reporting research results and findings obtained in the field of diabetes and covering a wide range of topics including risk factors for diabetes, diabetes complications, experimental diabetes mellitus, type 1 diabetes mellitus, type 2 diabetes mellitus, gestational diabetes, diabetic angiopathies, diabetic cardiomyopathies, diabetic coma, diabetic ketoacidosis, diabetic nephropathies, diabetic neuropathies, Donohue syndrome, fetal macrosomia, and prediabetic state.

INDEXING/ABSTRACTING

The *WJD* is now abstracted and indexed in Science Citation Index Expanded (SCIE, also known as SciSearch®), Current Contents/Clinical Medicine, Journal Citation Reports/Science Edition, PubMed, PubMed Central, Reference Citation Analysis, China Science and Technology Journal Database, and Superstar Journals Database. The 2024 Edition of Journal Citation Reports® cites the 2023 journal impact factor (JIF) for *WJD* as 4.2; JIF without journal self cites: 4.1; 5-year JIF: 4.2; JIF Rank: 40/186 in endocrinology and metabolism; JIF Quartile: Q1; and 5-year JIF Quartile: Q2.

RESPONSIBLE EDITORS FOR THIS ISSUE

NAME OF JOURNAL
World Journal of Diabetes

ISSN
ISSN 1948-9358 (online)

LAUNCH DATE
June 15, 2010

FREQUENCY
Monthly

EDITORS-IN-CHIEF
Lu Cai, Md. Shahidul Islam, Michael Horowitz

EDITORIAL BOARD MEMBERS
https://www.wjgnet.com/1948-9358/editorialboard.htm

PUBLICATION DATE
October 15, 2024

INSTRUCTIONS TO AUTHORS
https://www.wjgnet.com/bpg/gerinfo/204

GUIDELINES FOR ETHICS DOCUMENTS
https://www.wjgnet.com/bpg/GerInfo/287

GUIDELINES FOR NON-NATIVE SPEAKERS OF ENGLISH
https://www.wjgnet.com/bpg/gerinfo/240

PUBLICATION ETHICS
https://www.wjgnet.com/bpg/GerInfo/288

PUBLICATION MISCONDUCT
https://www.wjgnet.com/bpg/gerinfo/208

ARTICLE PROCESSING CHARGE
https://www.wjgnet.com/bpg/gerinfo/242

STEPS FOR SUBMITTING MANUSCRIPTS
https://www.wjgnet.com/bpg/GerInfo/239

ONLINE SUBMISSION
https://www.f6publishing.com

World Journal of Diabetes

Submit a Manuscript: https://www.f6publishing.com

World J Diabetes 2024 October 15; 15(10): 2152-2156

DOI: 10.4239/wjd.v15.i10.2152

ISSN 1948-9358 (online)

LETTER TO THE EDITOR

Gut microbiota modulating therapy for diabetes mellitus should be individualized

Jin Wang, Hong-Juan Wei, Rui-Feng Mao, Xin Chang

Specialty type: Endocrinology and metabolism

Provenance and peer review: Invited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's classification
Scientific Quality: Grade A
Novelty: Grade A
Creativity or Innovation: Grade A
Scientific Significance: Grade A

P-Reviewer: Liu J

Received: July 11, 2024
Revised: September 14, 2024
Accepted: September 18, 2024
Published online: October 15, 2024
Processing time: 76 Days and 22.4 Hours

Jin Wang, Xin Chang, Department of Ultrasound Medicine, Nanjing Lishui People's Hospital, Zhongda Hospital Lishui Branch, Southeast University, Nanjing 211200, Jiangsu Province, China

Hong-Juan Wei, Neonatal Intensive Care Unit, Nanjing Lishui People's Hospital, Zhongda Hospital Lishui Branch, Southeast University, Nanjing 211200, Jiangsu Province, China

Rui-Feng Mao, School of Life Science, Huaiyin Normal University, Huai'an 223300, Jiangsu Province, China

Co-first authors: Jin Wang and Hong-Juan Wei.

Corresponding author: Xin Chang, PhD, Chief Physician, Professor, Department of Ultrasound Medicine, Nanjing Lishui People's Hospital, Zhongda Hospital Lishui Branch, Southeast University, No. 86 Chongwen Road, Lishui District, Nanjing 211200, Jiangsu Province, China. changxinnj@163.com

Abstract

In this editorial, we commented on two articles published online in August and September 2024 in the *World Journal of Diabetes*, which focused on modifying the gut microbiota (GM) to prevent or delay the progression of diabetes mellitus (DM) and DM-related complications. Numerous studies, many of which are animal studies, have indicated the potential role of GM in the pathogenesis of DM. However, the detailed causality and mechanisms between GM and DM have not been fully clarified. Although there have been some reports of a potential role of modifying the GM in treating DM, most lack long-term observations and are not mechanistic. Additionally, the GM and its role in DM might vary among individuals; therefore, GM-targeted interventions should be individualized to realize their therapeutic potential.

Key Words: Diabetes mellitus; Gut microbiota; Dysbiosis; Causality; Individualized interventions

Core Tip: This editorial focuses on the research progress regarding the gut microbiota (GM) in the development of diabetes mellitus (DM). Revealing and understanding the precise causality and mechanisms between the GM and DM may facilitate investigations focused on modifying the GM to ameliorate DM and its complications. Additionally, as a result of the considerable interindividual heterogeneity in the GM, more precise and personalized GM-targeting therapeutic interventions should be considered.

Citation: Wang J, Wei HJ, Mao RF, Chang X. Gut microbiota modulating therapy for diabetes mellitus should be individualized. *World J Diabetes* 2024; 15(10): 2152-2156
URL: https://www.wjgnet.com/1948-9358/full/v15/i10/2152.htm
DOI: https://dx.doi.org/10.4239/wjd.v15.i10.2152

TO THE EDITOR

As a common metabolic and endocrine disease, diabetes mellitus (DM) is characterized by glycometabolism disorders resulting from malfunctions in insulin secretion and/or its action. The high and steadily increasing global prevalence of DM poses an increasing burden on health care, particularly in developed countries[1]. DM has various subtypes, the most important being type 1 DM (T1DM) and type 2 DM (T2DM), and more than 90% of DM cases worldwide are T2DM[2]. As a chronic autoimmune disease, T1DM is characterized by insulin deficiency resulting from inflammation and the destruction of insulin-producing pancreatic β-cells mediated by autoantibodies. Although T1DM can occur at any age, it primarily affects children and adolescents[3]. Individuals with T1DM require a lifelong dependence on exogenous insulin as there is no cure[3]. In contrast, T2DM mainly occurs due to insulin resistance, or insulin deficiency resulting from β-cell dysfunction caused by mechanisms other than the autoimmune process similar to T1DM[4]. As a result of its progressive nature, T2DM is treated by lifestyle changes, oral antidiabetic drugs, or insulin injections depending on its stage[5]. After the diagnosis of T1DM or T2DM, glycemic control is the central goal of DM management, as hyperglycemia contributes greatly to DM itself and to its various complications[6]. However, it is not easy to achieve and maintain optimal glycemic control, and only 25% of T2DM patients receiving insulin therapy achieve good glycemic control[7]. Thus, there is a considerable need to further improve the management of DM, which requires a comprehensive consideration of various aspects.

The optimal management of DM depends on a full understanding of its associated risk factors and pathogenesis. Various potential risk factors for developing DM, such as a family history of DM (genetics), altered and/or unhealthy lifestyle behaviors, obesity, and environmental irritants, have been suggested[8]. Scientific research has shown that the balance of the gut microbiota (GM) is closely related to the state of health, and the diversity, activity and composition changes of the GM, as an important part of this huge ecosystem, can significantly affect the metabolic function, and the immune response. Furthermore, gastrointestinal microflora has been shown to play a key role in maintaining homeostasis in, and GM dysbiosis might contribute to various diseases, including DM[9]. Therefore, GM might be an important environmental factor associated with the pathogenesis of DM. In the 8th[10] and 9th[11] issues of the *World Journal of Diabetes* this year, there were reports on the modification of the GM in the treatment of DM and DM-related complications. Inspired by these two papers, we wrote this editorial to describe the progress in research on modifying the GM in the treatment of DM.

As a large reservoir of microorganisms occupying up to 95% of the entire human microbiota, the GM mainly contains six phyla, of which *Firmicutes* and *Bacteroidetes* are the most dominant. In fact, an altered GM composition is an important characteristic of DM patients. In addition, increasing evidence indicates an underlying association between GM and DM [12,13]. Compared with healthy children, those with either new-onset T1DM or autoantibody positivity show GM dysbiosis, characterized by reduced diversity, reduced vitality and a lower ratio of *Firmicutes*-to-*Bacteroidetes*. Additionally, various specific gut microbiome changes have been observed in T1DM patients. These microbial changes might induce a proinflammatory environment and increase paracellular permeability, ultimately stimulating autoimmunity[13,14]. The occurrence of GM dysbiosis might contribute to the development and progression of T2DM *via* various mechanisms, such as increasing intestinal permeability by decreasing short-chain fatty acid (SCFA)-producing bacteria and modifying glucose homeostasis by altering bile acid signaling[15]. Therefore, it is generally believed that profiling the gut microbial composition and function in diagnosed or potential DM patients and then rebalancing or modifying gut dysbacteriosis could reverse DM or delay its development.

Under normal circumstances, as a dynamically changing microbial community, the complex gut microbial ecosystem can be affected by several factors, such as genetics, age, place of residence, diet, use of probiotics and prebiotics, medications, and environmental factors (*i.e.*, air pollution)[16]. Various strategies aimed at preventing or treating DM by modifying the GM have shown encouraging results, among which dietary intervention may be among the most effective, as dietary changes can easily affect the composition of the GM[17]. For example, a Mediterranean diet, which is rich in monounsaturated fats and fiber, could induce changes in the GM to increase SCFA production, especially butyrate, to exhibit immunoregulatory activities, suggesting a potential role in preventing or delaying the progression of T1DM and T1DM-related complications[18]. Currently, various studies have revealed that different dietary components may contribute to primary prevention, secondary prevention, tertiary prevention, or complication prevention in patients with T1DM[19]. However, whether these dietary interventions function *via* GM-mediated processes is unclear; and indeed, the

protective effects of some dietary interventions on T1DM are not mediated by GM[20]. There are more studies on the associations between diet and GM with T2DM than with T1DM, and strong associations have been reported[21]. Similarly, a Mediterranean diet improved glycemic control in patients with T2DM, possibly by increasing GM diversity and richness. Therefore, GM richness has potential as a biomarker for analyzing the efficacy of dietary interventions in T2DM patients[22]. A high-fiber diet resulted in increased levels of *Lactobacillus*, *Bifidobacterium* and *Akkermansia* and decreased levels of *Desulfovibrio*, *Klebsiella* and other opportunistic pathogens, thus leading to better glycemic control[23]. However, it should be noted that alterations in the GM induced by dietary changes seem temporary; therefore, long-term studies of dietary interventions that evaluate the microbiome and the quality of DM control are needed[24]. Future research will focus on developing diet plans rich in specific prebiotics, dietary fiber or oligosaccharides that promote the growth of beneficial bacteria and inhibit that of harmful bacteria, thereby optimizing the composition of the GM and improving the metabolic status of diabetic patients. Additionally, nutrition recommendations personalized according to the characteristics of the GM, such as by adjusting the diet plan, would likely achieve the best results. In addition to dietary interventions, numerous medications, especially antibiotics, can significantly impact or regulate GM homeostasis and could be used to modify the GM composition in patients with DM. Indeed, antidiabetic medications, such as metformin, might also act by regulating the GM balance[25]. Additionally, probiotics and prebiotics have been reported to ameliorate DM by affecting the GM[17,26,27].

As described above, various studies have investigated the association between the GM and DM and the effect of modifying the GM on the progression of DM and DM-related complications. However, it should be noted that most studies of the causality of the relationship between GM and DM used mouse models, and of the few studies conducted in human subjects, most were observational. In addition, human studies have focused on patients with one type of DM. In studies of T2DM, newly diagnosed patients are most frequently used, whereas studies of T1DM typically involve children with an unformed or unstable GM[28]. The reproducibility of human research might be poor due to variations in the types of interventions, geographic locations, participants, and study designs and settings. Moreover, some human studies have reported contradictory observations[17]. In other words, research on the association between the GM and DM is at a very early stage, and it is difficult to clarify the detailed causality of the relationship between a specific gut bacterium and phenotypic exposure; thus, more research is still required. This ambiguous association is a major obstacle to preventing or delaying the progression of DM and DM-related complications by modifying the GM. Future studies evaluate how the GM directly or indirectly participate in the regulation of blood glucose and the maintenance of insulin sensitivity through the production of SCFAs, the regulation of bile acid metabolism, and the release of inflammatory factor. An in-depth analysis of these mechanisms will provide a solid theoretical basis for the treatment of DM by modulating the intestinal flora.

Interindividual heterogeneity is another important factor that should be considered. Being influenced by various factors, the composition and richness of the GM are highly variable among individuals. In the future, DM treatment is likely to be personalized. Precise analysis of the GM through high-throughput sequencing technology can identify members of the microbiota or metabolites associated with the diabetes risk, enabling the design of targeted dietary interventions or probiotic/prebiotic supplementation programs to achieve optimal treatment outcomes. Therefore, given that the altered GM of patients with DM may be individual specific, analysis of the GM composition by cutting-edge technologies, such as next-generation sequencing is needed. However, despite the popularity of next-generation sequencing, the cost and time constraints preclude regular monitoring of changes in the GM. As an alternative, a multiplex TaqMan qPCR assay targeting various gut microbial phyla could be used to monitor alterations in the GM[29]. On the basis of the potential role of an altered GM in DM, individualized therapeutic interventions targeting the GM, such as personalized nutrition and/or probiotics or prebiotic mixtures, should be designed and evaluated for their ability to prevent or delay the progression of DM and DM-related complications. Moreover, the long-term effects and sustainability of these individualized therapeutic interventions should be investigated.

The implementation of GM-based treatment strategies for DM requires close collaboration among endocrinology, nutrition, microbiology, genetics and bioinformatics. Interdisciplinary research can provide insight into the pathogenesis of DM, thereby accelerating the development and application of new treatments. As data accumulate, GM regulation therapy is expected to be included in the clinical treatment guidelines for DM alongside drug therapy and lifestyle intervention. Moreover, governments and health institutions should strengthen relevant policies, encourage scientific research and innovation, promote the clinical translation of scientific research, and improve DM prevention and control.

CONCLUSION

In conclusion, various genetic and environmental factors contribute to the risk of DM. Accumulating evidence suggests that GM dysbiosis should be considered a causative environmental factor of T1DM and T2DM. The discovery of the relationship between the GM and DM opens a new horizon for the management and treatment of DM and heralds the arrival of a new era of DM control based on the regulation of the human microecology. However, the precise causality and mechanisms of the relationship between the GM and DM have not been revealed and deserve further elucidation, which will facilitate modification of the GM to ameliorate DM and its complications. Moreover, the role of the GM in DM might show interindividual heterogeneity, highlighting the importance of more precise and personalized GM-targeting therapeutic interventions. As research continues and technologies advance, we believe that further breakthroughs in this field will lead to the development of safer and more effective treatment options for DM.

FOOTNOTES

Author contributions: Wang J and Wei HJ conceptualized and drafted the manuscript; Mao RF edited the manuscript; Chang X did further revisions and editorial corrections before submission. All authors have read and agreed to the published version of this manuscript. Wang J and Wei HJ contributed equally to this work as co-first authors.

Supported by Medical Education Collaborative Innovation Fund of Jiangsu University, No. JDYY2023101.

Conflict-of-interest statement: Authors declare no conflict of interests for this article.

Country of origin: China

ORCID number: Jin Wang 0009-0008-7680-8547; Hong-Juan Wei 0000-0002-7371-6988; Rui-Feng Mao 0000-0001-7596-6713; Xin Chang 0000-0003-0277-7705.

S-Editor: Qu XL
L-Editor: A
P-Editor: Zhang L

REFERENCES

1 **Sun H**, Saeedi P, Karuranga S, Pinkepank M, Ogurtsova K, Duncan BB, Stein C, Basit A, Chan JCN, Mbanya JC, Pavkov ME, Ramachandaran A, Wild SH, James S, Herman WH, Zhang P, Bommer C, Kuo S, Boyko EJ, Magliano DJ. IDF Diabetes Atlas: Global, regional and country-level diabetes prevalence estimates for 2021 and projections for 2045. *Diabetes Res Clin Pract* 2022; **183**: 109119 [PMID: 34879977 DOI: 10.1016/j.diabres.2021.109119]

2 **da Silva A**, Caldas APS, Rocha DMUP, Bressan J. Triglyceride-glucose index predicts independently type 2 diabetes mellitus risk: A systematic review and meta-analysis of cohort studies. *Prim Care Diabetes* 2020; **14**: 584-593 [PMID: 32928692 DOI: 10.1016/j.pcd.2020.09.001]

3 **Quattrin T**, Mastrandrea LD, Walker LSK. Type 1 diabetes. *Lancet* 2023; **401**: 2149-2162 [PMID: 37030316 DOI: 10.1016/S0140-6736(23)00223-4]

4 **Galicia-Garcia U**, Benito-Vicente A, Jebari S, Larrea-Sebal A, Siddiqi H, Uribe KB, Ostolaza H, Martín C. Pathophysiology of Type 2 Diabetes Mellitus. *Int J Mol Sci* 2020; **21** [PMID: 32872570 DOI: 10.3390/ijms21176275]

5 **Landgraf R**, Aberle J, Birkenfeld AL, Gallwitz B, Kellerer M, Klein H, Müller-Wieland D, Nauck MA, Reuter HM, Siegel E. Therapy of Type 2 Diabetes. *Exp Clin Endocrinol Diabetes* 2019; **127**: S73-S92 [PMID: 31860927 DOI: 10.1055/a-1018-9106]

6 **Yang T**, Qi F, Guo F, Shao M, Song Y, Ren G, Linlin Z, Qin G, Zhao Y. An update on chronic complications of diabetes mellitus: from molecular mechanisms to therapeutic strategies with a focus on metabolic memory. *Mol Med* 2024; **30**: 71 [PMID: 38797859 DOI: 10.1186/s10020-024-00824-9]

7 **Pitak P**, Tasai S, Kumpat N, Na Songkla P, Fuangchan A, Krass I, Dhippayom T. The prevalence of glycemic control in patients with type 2 diabetes treated with insulin: a systematic review and meta-analysis. *Public Health* 2023; **225**: 218-228 [PMID: 37939463 DOI: 10.1016/j.puhe.2023.10.015]

8 **Chan JC**, Malik V, Jia W, Kadowaki T, Yajnik CS, Yoon KH, Hu FB. Diabetes in Asia: epidemiology, risk factors, and pathophysiology. *JAMA* 2009; **301**: 2129-2140 [PMID: 19470990 DOI: 10.1001/jama.2009.726]

9 **Zhao M**, Chu J, Feng S, Guo C, Xue B, He K, Li L. Immunological mechanisms of inflammatory diseases caused by gut microbiota dysbiosis: A review. *Biomed Pharmacother* 2023; **164**: 114985 [PMID: 37311282 DOI: 10.1016/j.biopha.2023.114985]

10 **Li YY**, Guan RQ, Hong ZB, Wang YL, Pan LM. Advances in the treatment of diabetic peripheral neuropathy by modulating gut microbiota with traditional Chinese medicine. *World J Diabetes* 2024; **15**: 1712-1716 [PMID: 39192853 DOI: 10.4239/wjd.v15.i8.1712]

11 **Qureshi W**, Dar MA, Rather MY. New therapy for metabolic syndrome: Gut microbiome supplementation. *World J Diabetes* 2024; **15**: 1833-1836 [PMID: 39280175 DOI: 10.4239/wjd.v15.i9.1833]

12 **Zhang H**, Ma L, Peng W, Wang B, Sun Y. Association between gut microbiota and onset of type 2 diabetes mellitus: a two-sample Mendelian randomization study. *Front Cell Infect Microbiol* 2024; **14**: 1327032 [PMID: 38596649 DOI: 10.3389/fcimb.2024.1327032]

13 **Del Chierico F**, Rapini N, Deodati A, Matteoli MC, Cianfarani S, Putignani L. Pathophysiology of Type 1 Diabetes and Gut Microbiota Role. *Int J Mol Sci* 2022; **23** [PMID: 36498975 DOI: 10.3390/ijms232314650]

14 **Ismail HM**, Evans-Molina C. Does the Gut Microbiome Play a Role in Obesity in Type 1 Diabetes? Unanswered Questions and Review of the Literature. *Front Cell Infect Microbiol* 2022; **12**: 892291 [PMID: 35873174 DOI: 10.3389/fcimb.2022.892291]

15 **Borges LP**, de Jesus PC, de Souza JB, Silva DMRR, Moura PHM, Santos RS, Barreto MDS, Guimarães AG, Santana LADM, Cabral-Marques O, Silva EED. The influence of gut microbiota on the progression of Type 2 Diabetes: a new perspective for treatment and prevention. *EXCLI J* 2024; **23**: 629-630 [PMID: 38887397 DOI: 10.17179/excli2024-7036]

16 **Anwar H**, Iftikhar A, Muzaffar H, Almatroudi A, Allemailem KS, Navaid S, Saleem S, Khurshid M. Biodiversity of Gut Microbiota: Impact of Various Host and Environmental Factors. *Biomed Res Int* 2021; **2021**: 5575245 [PMID: 34055983 DOI: 10.1155/2021/5575245]

17 **Li WZ**, Stirling K, Yang JJ, Zhang L. Gut microbiota and diabetes: From correlation to causality and mechanism. *World J Diabetes* 2020; **11**: 293-308 [PMID: 32843932 DOI: 10.4239/wjd.v11.i7.293]

18 **Calabrese CM**, Valentini A, Calabrese G. Gut Microbiota and Type 1 Diabetes Mellitus: The Effect of Mediterranean Diet. *Front Nutr* 2020;

7: 612773 [PMID: 33521039 DOI: 10.3389/fnut.2020.612773]

19 **Neuman V**, Plachy L, Pruhova S, Sumnik Z. Dietary Components in the Pathogenesis and Prevention of Type 1 Diabetes in Children. *Horm Res Paediatr* 2024; 1-11 [PMID: 38838652 DOI: 10.1159/000539575]

20 **Neuman V**, Pruhova S, Kulich M, Kolouskova S, Vosahlo J, Romanova M, Petruzelkova L, Havlik J, Mascellani A, Henke S, Sumnik Z, Cinek O. Changes in the gut bacteriome upon gluten-free diet intervention do not mediate beta cell preservation. *Diabetologia* 2023; **66**: 241-246 [PMID: 36194251 DOI: 10.1007/s00125-022-05805-3]

21 **Farahbod K**, Slouha E, Gerts A, Rezazadah A, Clunes LA, Kollias TF. The Effects of Diet Intervention on the Gut Microbiota in Type 2 Diabetes Mellitus: A Systematic Review. *Cureus* 2024; **16**: e56737 [PMID: 38646363 DOI: 10.7759/cureus.56737]

22 **Ismael S**, Silvestre MP, Vasques M, Araújo JR, Morais J, Duarte MI, Pestana D, Faria A, Pereira-Leal JB, Vaz J, Ribeiro P, Teixeira D, Marques C, Calhau C. A Pilot Study on the Metabolic Impact of Mediterranean Diet in Type 2 Diabetes: Is Gut Microbiota the Key? *Nutrients* 2021; **13** [PMID: 33917736 DOI: 10.3390/nu13041228]

23 **Chen L**, Liu B, Ren L, Du H, Fei C, Qian C, Li B, Zhang R, Liu H, Li Z, Ma Z. High-fiber diet ameliorates gut microbiota, serum metabolism and emotional mood in type 2 diabetes patients. *Front Cell Infect Microbiol* 2023; **13**: 1069954 [PMID: 36794003 DOI: 10.3389/fcimb.2023.1069954]

24 **Leeming ER**, Johnson AJ, Spector TD, Le Roy CI. Effect of Diet on the Gut Microbiota: Rethinking Intervention Duration. *Nutrients* 2019; **11** [PMID: 31766592 DOI: 10.3390/nu11122862]

25 **Xi Y**, Xu PF. Diabetes and gut microbiota. *World J Diabetes* 2021; **12**: 1693-1703 [PMID: 34754371 DOI: 10.4239/wjd.v12.i10.1693]

26 **Megur A**, Daliri EB, Baltriukienė D, Burokas A. Prebiotics as a Tool for the Prevention and Treatment of Obesity and Diabetes: Classification and Ability to Modulate the Gut Microbiota. *Int J Mol Sci* 2022; **23** [PMID: 35682774 DOI: 10.3390/ijms23116097]

27 **Song H**, Xue H, Zhang Z, Wang J, Li A, Zhang J, Luo P, Zhan M, Zhou X, Chen L, Fang Y. Amelioration of Type 2 Diabetes Using Four Strains of Lactobacillus Probiotics: Effects on Gut Microbiota Reconstitution-Mediated Regulation of Glucose Homeostasis, Inflammation, and Oxidative Stress in Mice. *J Agric Food Chem* 2023; **71**: 20801-20814 [PMID: 37991826 DOI: 10.1021/acs.jafc.3c04665]

28 **Salamon D**, Sroka-Oleksiak A, Kapusta P, Szopa M, Mrozińska S, Ludwig-Słomczyńska AH, Wołkow PP, Bulanda M, Klupa T, Małecki MT, Gosiewski T. Characteristics of gut microbiota in adult patients with type 1 and type 2 diabetes based on nextgeneration sequencing of the 16S rRNA gene fragment. *Pol Arch Intern Med* 2018; **128**: 336-343 [PMID: 29657308 DOI: 10.20452/pamw.4246]

29 **Jo YJ**, Tagele SB, Pham HQ, Jung Y, Ibal JC, Choi S, Kang GU, Park S, Kang Y, Kim S, Koh H, Shin JH. In Situ Profiling of the Three Dominant Phyla Within the Human Gut Using TaqMan PCR for Pre-Hospital Diagnosis of Gut Dysbiosis. *Int J Mol Sci* 2020; **21** [PMID: 32168885 DOI: 10.3390/ijms21061916]

Published by **Baishideng Publishing Group Inc**
7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA
Telephone: +1-925-3991568
E-mail: office@baishideng.com
Help Desk: https://www.f6publishing.com/helpdesk
https://www.wjgnet.com