DIET AND THE MICROBIOME: INTERACTION PATHWAYS IN CHRONIC INTESTINAL DISEASES (LITERATURE REVIEW)

Vasylyeva I.M., Nakonechna O.A., Makieieva N.I., Yarmysh N.V., Denysenko S.A.

Kharkiv National Medical University, Kharkiv, Ukraine

https://doi.org/10.35339/ic.2026.13.1.vnm

ABSTRACT

Background. Nutrition is the science of food, nutrients, their effects, interactions, and balance in the context of human health and disease. Nutraceuticals provide the body with energy substrates, which are converted in the body's cells into important metabolites used in physiological and biochemical processes. Diet plays an important role in shaping the qualitative and quantitative composition of the human gut microbiome.

Aim. To analyze the interaction between diet and the gut microbiome, and to investigate the role of specific nutrients in the development and progression of chronic intestinal diseases.

Materials and Methods. The study was carried out using the bibliosemantic method and system analysis. For analysis, the sources of 2012–2026 were selected. from scientometric databases PubMed, Scopus, ScienceDirect, EMBASE, Medline, Cochrane Library and Google Scholar. The study was carried out as a private initiative of the authors without grant support and state registration of the topic.

Research Ethics. For the analysis, only those sources were selected, the authors of which adhered to modern bioethical norms when performing their research.

Results. Fermentation of dietary fiber by the colonic gut microbiota produces short-chain fatty acids, which provide [60–70]% of the energy needs of colonocytes, maintain intestinal barrier integrity, and modulate cell proliferation. Polysaccharide deficiency leads to destruction of the mucus layer, erosions, and "leaky gut" syndrome. High consumption of fruit, vegetables, legumes and potatoes reduces the risk of Crohn's disease (adjusted Hazard Ratio (aHR) = 0.44) but does not affect ulcerative colitis; excessive potato consumption increases the risk of ulcerative colitis (aHR=1.51). Insoluble dietary fiber may provoke abdominal pain, diarrhea and flatulence, whereas soluble fiber stimulates *Lactobacillus sp*. and *Bifidobacteria sp.* Legumes, in particular *Phaseolus vulgaris Navy bean*, due to their content of lysine, iron, phosphatidylserine, apigenin and coumaric acid, reduce oxidative stress, inflammation and the risk of colorectal adenoma recurrence.

Conclusions. The interaction between diet and the microbiome through fermentation of dietary fiber is a key mechanism for maintaining intestinal homeostasis. Different dietary components have a differentiated effect on the risk and course of Crohn's disease and ulcerative colitis. Unbalanced nutrition causes dysbiosis, increased intestinal barrier permeability and chronic inflammation, which justifies the need for personalized dietary approaches.

Keywords: *nutrition, dietary fiber, inflammatory intestinal diseases*.

Introduction

In the modern world, one of the key components of public health care is nutriology. According to the World Health Organization, nutriology is the science of nutrition, nutrients and substances contained in food nutrients; their action, interaction and balance in relation to human health and disease; as well as the processes by which the body digests, absorbs, transports, uses and excretes metabolic end products [1].

Nutrients provide the body with energy substrates, which are converted in the body's cells into important metabolites used in physiological and biochemical processes [2]. The quality of nutrition plays an important role in the development and course of chronic non-communicable diseases, including cardiovascular diseases [3; 4], diabetes [5], obesity [6], intestinal inflammatory processes, and some types of cancer [7]. These diseases share common risk factors, one of which is improper or unbalanced diet [8]. Non-communicable diseases are associated not only with diet and lifestyle but also with age-related characteristics and biological

Corresponding Author:
Vasylyeva Iryna M. – Candidate of Biological Sciences, Docent, Associate Professor of the Department of Biological Chemistry, Kharkiv National Medical University, Ukraine.
✉ 4, Nauky ave., Kharkiv, 61022, Ukraine.
E-mail: im.vasylieva@knmu.edu.ua

aging processes [9], manifested by increased oxidative stress, mitochondrial dysfunction, cell aging [10–12], changes in neuroendocrine signaling and activity of certain enzymes. Some experimental studies have demonstrated a modulating effect of dietary therapy on diseases caused by metabolic disorders [13].

Currently, individual approaches in personalized diet are being considered, focusing on genetic characteristics related to certain foods [14], on the state of the gut microbiome and the processes of digestion in the gastrointestinal tract [15; 16], and on the immune system's response to food agents [17]. Current research indicates that food preferences are closely linked to the development and severity of diseases. These findings highlight the importance of developing nutritional programs to resolve or alleviate the course of many diseases [18].

Thus, diet plays an important role in shaping the qualitative and quantitative composition of the human gut microbiome [19; 20]. In the current scientific literature, there is growing interest in the influence of various diets on the state of the microbiome, as well as on the improvement of intestinal condition in inflammation, ulcerative colitis and Crohn's disease [19; 21].

The **aim** of the study was to analyze of the interaction between diet and the gut microbiome, and to investigate the role of specific nutrients in the development and progression of chronic intestinal diseases.

Materials and Methods

The study was conducted using the bibliosemantic method and systemic analysis. Sources from 2012–2026 indexed in the scientometric databases PubMed, Scopus, ScienceDirect, EMBASE, Medline, Cochrane Library and Google Scholar were selected for analysis using the keywords: diet, gut microbiome, inflammatory bowel disease, irritable bowel syndrome, dietary fiber, short-chain fatty acids, intestinal barrier, dysbiosis, nutraceuticals, chronic intestinal inflammation, personalized nutrition, microbiota-targeted diet. The selection of sources was carried out in two stages: primary screening by titles and abstracts (including experimental studies, clinical trials and reviews) followed by full-text analysis. Exclusion criteria were abstracts, case reports, and topic-irrelevant publications

Research Ethics

This work is a review of literary sources that are publicly available. No patients were involved in the study, which does not require additional approval from an ethics committee. Only those sources whose authors adhered to modern bioethical standards when conducting their research were selected for analysis.

Results

In the gastrointestinal tract, the processes of digestion and absorption of nutrients take place. Clinical trials and epidemiological studies confirm the influence of specific dietary components, such as probiotics, polyphenols, omega-3 fatty acids, and vitamins, on the growth and composition of the gut microbiota, which generates various biochemical metabolites that positively or negatively affect the mucous membrane of the intestinal epithelium [22; 23]. Regular consumption of probiotics helps strengthen the intestinal barrier and reduces the risk of inflammatory bowel diseases. Polyphenols and omega-3 fatty acids have an anti-inflammatory effect, lower the level of cholesterol of low-density lipoproteins, improve insulin sensitivity and reduce oxidative stress [22]. In a healthy intestine, about 5% of primary bile acids undergo bacterial biotransformation, and the composition of the bile acid pool is closely related to the structure of the microbial community. Disturbance of this interaction is observed in Crohn's disease, ulcerative colitis and metabolic syndrome [23].

The contents of the intestinal lumen do not penetrate into the bloodstream due to the presence of epithelial cells. Seven types of epithelial cells are known, represented by enterocytes, goblet cells, Paneth cells, microfold cells, enteroendocrine cells, cup cells and tuft cells, which form both the biological barrier (represented by the gut microbiota) and the mechanical barrier (represented by the intestinal epithelium itself) of the body [24]. Epithelial Cells of the Intestine (ECI) are the first to respond to changes in the composition of the microbiota in non-infectious inflammatory bowel diseases. As a result of disruption of the intestinal barrier, destruction of tight intercellular protein contacts and translocation of microbial products, the type of metabolism in ECI changes [25]. The epithelial layer is part of the innate immunity, recognizing pathogenic microorganisms, and is also a source of mucin, antimicrobial peptides, cytokines and chemokines [26].

The inner mucous layer of the large intestine is constantly renewed under physiological conditions. However, under certain conditions, in response to factors that cause destruction or increased permeability of the intestinal mucous layer, bacteria reach the epithelial cells and cause inflam-

matory reactions, which are a common mechanism in all experimental models and in patients with non-infectious intestinal diseases [27].

In a healthy intestine, there is a balance between commensal microbiota and the mucous layer. Polysaccharides serve as the main source of nutrition for the microbiota, allowing the preservation of mucin-degrading bacteria and maintaining homeostasis. A diet with a reduced amount of polysaccharides leads to a loss of part of the bacteria's nutritional potential. The latter begin to break down the mucus layer (mucin) on the intestinal surface, using it as an alternative source of carbohydrates. The consequence of these events may be the appearance of erosions [28; 29] and the formation of a condition known as "leaky gut" [30]. Okada T. et al. (2013) [31] showed that a high level of lactate produced by *Lactobacillus murinus* in the large intestine of mice that were initially starved and then switched to standard feeding accelerates the renewal of colonic epithelial cells (increased proliferation of enterocytes), however, these cells exhibited increased sensitivity to carcinogens, specifically the formation of a larger number of aberrant crypts. In the large intestine, during fermentation of fiber by the gut microbiota, Short-Chain Fatty Acids (SCFAs) (acetic, propionic and butyric acids) are formed. Other short-chain fatty acids, such as valerate, caproate and isovalerate, are produced in negligible amounts [32]. The total concentration of SCFAs in the proximal large intestine is [70–140] mM, and the molar ratio of acetate, propionate and butyrate is approximately 60:20:20 [6; 16; 32]. These fatty acids are used not only as an energy source (they provide [60–70]% of the energy needs of colonocytes [32]), but also as modulators of the physiological function of enteric epithelial cells and immune cells [33]. For example, butyrate promotes cell proliferation and acts as an inducer of the cell cycle of ECI [34].

Dietary fiber – a complex of biopolymers that form the walls of plant cells – can be divided into dietary fiber that is partially fermented by bacteria, non-fermented dietary fiber, and dietary fiber that is completely fermented by bacteria [35; 36]. The World Health Organization recommends consuming up to 5 servings of vegetables and fruit per day, which amounts to 400 g (of which [3–4] servings of vegetables, corresponding to [240–320] g, and [1–2] servings of fruit, corresponding to [80–160] g). This ensures normal body functioning and reduces the risks and progression of diseases [37]. However, for However, people with irritable bowel syndrome are advised to reduce their servings to 3, because vegetables and fruit can cause various symptom complexes [38], depending on the type and amount of dietary fiber in the diet.

Meyer A. et al. (2026) [39] in a prospective study involving 341,519 individuals (the median follow-up period was 13.4 years) assessed the risks of developing Crohn's Disease (CD) and Ulcerative Colitis (UC) depending on the consumption of fruit, vegetables, legumes and potatoes (*Fig.*). It was found that high consumption of the combined group of these products was associated with a reduced risk of CD (Adjusted Risk Ratio (ARR) = 0.44; 95% Confidence Interval (CI) [0.26÷0.76]; p<0.01),

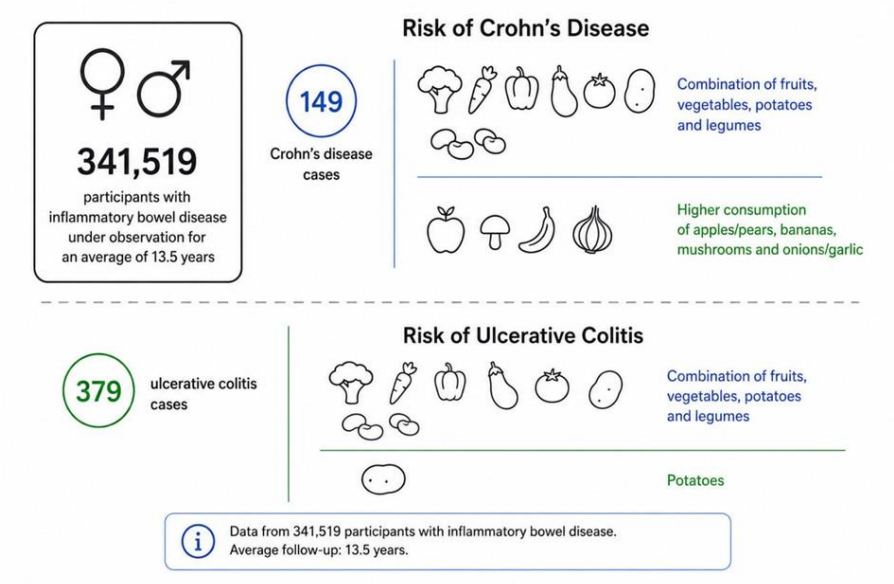

Fig. Dietary composition and the risk of developing inflammatory bowel disease [39].

but did not affect the risk of UC. At the same time, high consumption of potatoes was associated with an increased risk of UC (ARR=1.51; 95% CI [1.05÷2.17]; p<0.01). In particular, consumption of apples or pears; bananas; mushrooms; onions or garlic reduced the risk of CD.

In the current scientific literature, there are conflicting data regarding the role of insoluble dietary fiber. Some authors point to a positive effect, as it promotes water binding, increased stool volume and intestinal peristalsis. Other authors believe that the consumption of dietary fiber may worsen the condition in people with inflammatory bowel diseases and may cause abdominal pain, diarrhea, flatulence [40]. As for soluble fiber, it can directly or indirectly affect the microbiota, stimulating the colonization of *Lactobacillus sp.* and *Bifidobacteria sp.* [41].

It is known that in people with intestinal diseases, some cereal products can provoke or exacerbate the disease. Although cereal products are energy substrates, patients with inflammatory processes and irritable bowel are often advised to exclude foods rich in gluten from their diet, as it causes intestinal discomfort and may affect the course of the disease [42]. According to the national recommendations of various countries regarding the nutrition of patients with sensitive bowels, products based on wheat flour and other grain products containing insoluble dietary fiber should be excluded from the diet [43].

Legumes are one of the controversial inclusions in the diet of patients with inflammatory bowel diseases. It is known that common bean (*Phaseolus vulgaris*) contains phenolic compounds, dietary fiber, starch and proteins. Under the action of the gut microbiota, fermentation of legume components occurs and the formation of biologically active substances, SCFAs, which improve the condition of the intestine [44]. Phenolic compounds formed during fermentation can modulate the integrity of the intestinal mucosa and reduce oxidative stress, improving the condition in inflammatory bowel diseases, including ulcerative colitis. According to current literature, legumes of the *Phaseolus vulgaris* type, *Navy bean*, differ from other types in their high content of lysine, iron, phosphatidylserine, apigenin, coumaric acid, which is of certain importance for the gut microbiome [45]. Studies adding *Navy bean* to a high-fat diet showed an improvement in the inflammatory state of the intestine and a reduction in the degree of obesity. Clinical studies prove that bean consumption improves the state of the cardiovascular system [46] and reduces the risk of colorectal adenoma recurrence [47].

Discussion

The presented literature review confirms that diet is one of the main factors shaping the quantitative and qualitative composition of the gut microbiota, which, in turn, plays a decisive role in maintaining the homeostasis of the intestinal mucosa. The key mechanism linking dietary habits and the functioning of the gut microbiota is the fermentation of dietary fiber with the formation of SCFAs – acetate, propionate and butyrate [32; 33]. Butyrate, in particular, acts not only as the main energy substrate for colonocytes (providing [60–70]% of their energy needs), but also as a powerful modulator of proliferation and inflammation [32; 34]. Reduced consumption of polysaccharides leads to depletion of the nutritional potential of bacteria, forcing them to destroy the intestinal mucus layer, contributing to the appearance of erosions and the formation of leaky gut syndrome [28–30]. This creates a basis for chronic inflammation, especially in patients with Inflammatory Bowel Diseases (IBD).

At the same time, there are conflicting data in the current scientific literature regarding the role of insoluble dietary fiber. Although they promote water binding, increased stool volume and enhanced peristalsis, in patients with IBD their excessive consumption may provoke abdominal pain, diarrhea and flatulence [40]. This indicates the need for an individual approach to dietary prescription depending on the activity and phase of the disease.

Great interest is raised by the results of the large prospective study by Meyer A. et al. (2026) [39], which clearly demonstrated the differentiated effect of individual groups of plant foods on the risk of developing Crohn's Disease (CD) and Ulcerative Colitis (UC). High consumption of fruits, vegetables, legumes and potatoes in combination was associated with a reduced risk of CD (ARR=0.44), but did not affect UC. In contrast, excessive potato consumption was associated with an increased risk of UC (ARR=1.51). These data indicate the need not only for personalized, but also for nosology-specific dietary recommendations for patients with different forms of IBD.

Legumes deserve special attention, particularly the common bean (*Phaseolus vulgaris*) of the *Navy bean* type. The rich composition of these legumes (lysine, iron, phosphatidylserine, apigenin, coumaric acid) determines their protective effect on the intestinal mucosa, reduction of oxi-

dative stress and improvement of metabolic parameters [44; 45]. Experimental studies have shown that adding *Navy beans* to a high-fat diet reduces the severity of inflammation and obesity, and clinical data indicate a reduced risk of colorectal adenoma recurrence and improvement of the cardiovascular system [46; 47]. However, most of these data have been obtained from animal models or individual cohorts, which requires confirmation in randomized clinical trials.

An important aspect that lacks a clear answer is the role of gluten and cereal products in patients with IBD and irritable bowel syndrome. Although many national guidelines recommend excluding products made from wheat flour and other grains containing insoluble fiber [43], the evidence base regarding the appropriateness of a gluten-free diet in IBD remains limited and controversial. This issue requires further study, especially considering the possibility of cross-reactions and individual sensitivity.

The ***limitations*** of this review are, firstly, the heterogeneity of the designs of the included studies – from experimental animal studies to large cohort studies. Secondly, most dietary interventions were not standardized in terms of composition, duration and cooking methods. Thirdly, possible publication bias (the tendency to publish positive results) could have affected the generalizations. Finally, many studies did not take into account the genetic polymorphism of patients, which modulates their metabolic response to food nutrients.

Conclusions

1. The interaction between diet and the gut microbiome is realized through the fermentation of dietary fiber with the formation of short-chain fatty acids, which provide the energy needs of colonocytes ([60–70]%), modulate cell proliferation and maintain the integrity of the intestinal barrier. A deficiency of polysaccharides in the diet leads to depletion of the microbiota, destruction of the mucus layer, the appearance of erosions and "leaky gut" syndrome.

2. Different components of the diet have varying effects on the risk of developing and the course of inflammatory bowel diseases. High consumption of fruits, vegetables, legumes and potatoes is associated with a reduced risk of Crohn's disease, while excessive potato consumption increases the risk of ulcerative colitis. Insoluble dietary fiber may worsen the condition of patients (abdominal pain, diarrhea, flatulence), whereas soluble fiber stimulates the colonization of *Lactobacillus sp*. and *Bifidobacteria sp.*

3. Legumes, in particular *Phaseolus vulgaris* of the *Navy bean* type, due to their content of lysine, iron, phosphatidylserine, apigenin and coumaric acid, exhibit a protective effect on the intestinal mucosa, reduce oxidative stress and inflammation, and also lower the risk of colorectal adenoma recurrence.

4. Disruption of the diet-microbiome interaction caused by unbalanced nutrition leads to dysbiosis, increased intestinal barrier permeability and activation of chronic inflammation, which is a common pathogenetic mechanism in Crohn's disease, ulcerative colitis and irritable bowel syndrome. Personalized dietary approaches that take into account genetic characteristics, microbiome composition and disease stage are a promising direction for reducing the frequency of relapses and the risk of developing these pathologies.

Prospects for Further Research

Despite significant progress in understanding the diet-microbiome-chronic intestinal disease interaction, a number of key issues remain unresolved. Promising directions for further research include the development of individualized dietary approaches that take into account the patient's genetic characteristics, microbiome composition, nutritional status and disease phase, as well as the conduct of long-term randomized controlled trials.

Funding and Acknowledgments

The study was carried out as a private initiative of the authors without grant support and state registration of the topic.

Declarations

Conflict of interest is absent.

The authors confirm that during the preparation, writing and editing of this manuscript, generative Artificial Intelligence (AI) tools were used only within the limits permitted by the Generative AI Delegation Taxonomy (GAIDeT, 2025). Specifically, the latest version of ChatGPT (OpenAI, USA) was used to create the figure, and the current version of DeepSeek (the conversational AI assistant) was used to verify the quality of the translation. All other stages of the work (from the formation of the scientific idea to the final revision of the text) were carried out by the authors personally without the involvement of generative AI technologies.

Authors' Contributions

Contribution / Authors	A	B	C	D	E	F
Vasylyeva I.M.	+	+	+	+	+	+
Nakonechna O.A.	+			+	+	+
Makieieva N.I.	+			+	+	+
Yarmysh N.V.	+	+	+		+	+
Denysenko S.A.	+	+				+

Notes: A – concept; B – design; C – data collection;
D – statistical processing and interpretation of data;
E – writing or critical editing of the article;
F – approval of the final version for publication and agreement to be responsible for all aspects of the work.

References

1. Volkert D, Delzenne N, Demirkan K, Schneider S, Abbasoglu O, Bahat G, et al. Nutrition for the older adult – Current concepts. Report from an ESPEN symposium. Clin Nutr. 2024;43(8):1815-24. DOI: 10.1016/j.clnu.2024.06.020. PMID: 38970937.

2. Lyons CL, Roche HM. Nutritional Modulation of AMPK-Impact upon Metabolic-Inflammation. Int J Mol Sci. 2018;19(10):3092. DOI: 10.3390/ijms19103092. PMID: 30304866.

3. Liu D, Aziz NA, Pehlivan G, Breteler MMB. Cardiovascular correlates of epigenetic aging across the adult lifespan: a population-based study. Geroscience. 2023;45(3):1605-18. DOI: 10.1007/s11357-022-00714-0. PMID: 36752898.

4. Nyúl-Tóth Á, Patai R, Csiszar A, Ungvari A, Gulej R, Mukli P, et al. Linking peripheral atherosclerosis to blood-brain barrier disruption: elucidating its role as a manifestation of cerebral small vessel disease in vascular cognitive impairment. Geroscience. 2024;46(6):6511-36. DOI: 10.1007/s11357-024-01194-0. PMID: 38831182.

5. Wikström Shemer D, Mostafaei S, Tang B, Pedersen NL, Karlsson IK, Fall T, Hägg S. Associations between epigenetic aging and diabetes mellitus in a Swedish longitudinal study. Geroscience. 2024;46(5): 5003-14. DOI: 10.1007/s11357-024-01252-7. PMID: 38937415.

6. Cao X, Wang M, Zhou M, Mi Y, Fazekas-Pongor V, Major D, et al. Trends in prevalence, mortality, and risk factors of dementia among the oldest-old adults in the United States: the role of the obesity epidemic. Geroscience. 2024;46(5):4761-78. DOI: 10.1007/s11357-024-01180-6. PMID: 38696055.

7. Ungvari Z, Fekete M, Varga P, Lehoczki A, Fekete JT, Ungvari A, Győrffy B. Overweight and obesity significantly increase colorectal cancer risk: a meta-analysis of 66 studies revealing a 25–57% elevation in risk. Geroscience. 2025;47(3):3343-64. DOI: 10.1007/s11357-024-01375-x. PMID: 39379738.

8. Palliyaguru DL, Rudderow AL, Sossong AM, Lewis KN, Younts C, Pearson KJ, et al. Perinatal diet influences health and survival in a mouse model of leukemia. Geroscience. 2020;42(4):1147-55. DOI: 10.1007/s11357-020-00199-9. PMID: 32394346.

9. Marcozzi S, Bigossi G, Giuliani ME, Giacconi R, Piacenza F, Cardelli M, et al. Cellular senescence and frailty: a comprehensive insight into the causal links. Geroscience. 2023;45(6):3267-305. DOI: 10.1007/s11357-023-00960-w. PMID: 37792158.

10. Liu W, Wang J, Wang M, Hou H, Ding X, Ma L, Liu M. Oxidative Stress Factors Mediate the Association Between Life's Essential 8 and Accelerated Phenotypic Aging: NHANES 2005–2018. J Gerontol A Biol Sci Med Sci. 2024;79(1):glad240. DOI: 10.1093/gerona/glad240. PMID: 37813096.

11. Lopez FV, O'Shea A, Huo Z, DeKosky ST, Trouard TP, Alexander GE, et al. Neurocognitive correlates of cerebral mitochondrial function and energy metabolism using phosphorus magnetic resonance spectroscopy in older adults. Geroscience. 2025;47(2):2223-34. DOI: 10.1007/s11357-024-01403-w. PMID: 39477865.

12. Stankovics L, Ungvari A, Fekete M, Nyul-Toth A, Mukli P, Patai R, et al. The vasoprotective role of IGF-1 signaling in the cerebral microcirculation: prevention of cerebral microhemorrhages in aging. Geroscience. 2025;47(1):445-55. DOI: 10.1007/s11357-024-01343-5. PMID: 39271571.

13. Blanchard T, Eppe J, Mugnier A, Delfour F, Meynadier A. Enhancing cognitive functions in aged dogs and cats: a systematic review of enriched diets and nutraceuticals. Geroscience. 2025;47(3):2925-47. DOI: 10.1007/s11357-025-01521-z. PMID: 39827310.

14. Collier R. The DNA-based diet. CMAJ. 2017;189(1):E40-1. DOI: 10.1503/cmaj.109-5352. PMID: 27873755.

15. Yatsunenko T, Rey FE, Manary MJ, Trehan I, Dominguez-Bello MG, Contreras M, et al.Human gut microbiome viewed across age and geography. Nature. 2012;486(7402):222-7. DOI: 10.1038/nature11053. PMID: 22699611.

16. Rivera-Pinto J, Egozcue JJ, Pawlowsky-Glahn V, Paredes R, Noguera-Julian M, Calle ML. Balances: a New Perspective for Microbiome Analysis. mSystems. 2018;3(4):e00053-18. DOI: 10.1128/mSystems.00053-18. PMID: 30035234.

17. Zeng Q, Dong SY, Wu LX, Li H, Sun ZJ, Li JB, et al. Variable food-specific IgG antibody levels in healthy and symptomatic Chinese adults. PLoS One. 2013;8(1):e53612. DOI: 10.1371/journal.pone.0053612. PMID: 23301096.

18. Mozaffarian D. Dietary and Policy Priorities for Cardiovascular Disease, Diabetes, and Obesity: A Comprehensive Review. Circulation. 2016;133(2):187-225. DOI: 10.1161/CIRCULATIONAHA.115.018585. PMID: 26746178.

19. Wastyk HC, Fragiadakis GK, Perelman D, Dahan D, Merrill BD, Yu FB, et al. Gut-microbiota-targeted diets modulate human immune status. Cell. 2021;184(16):4137-53.e14. DOI: 10.1016/j.cell.2021.06.019. PMID: 34256014.

20. Deehan EC, Yang C, Perez-Muñoz ME, Nguyen NK, Cheng CC, Triador L, et al. Precision Microbiome Modulation with Discrete Dietary Fiber Structures Directs Short-Chain Fatty Acid Production. Cell Host Microbe. 2020;27(3):389-404.e6. DOI: 10.1016/j.chom.2020.01.006. PMID: 32004499.

21. Sugihara K, Kamada N. Diet-Microbiota Interactions in Inflammatory Bowel Disease. Nutrients. 2021;13(5):1533. DOI: 10.3390/nu13051533. PMID: 34062869.

22. Fekete M, Lehoczki A, Kryczyk-Poprawa A, Zábó V, Varga JT, Bálint M, et al. Functional Foods in Modern Nutrition Science: Mechanisms, Evidence, and Public Health Implications. Nutrients. 2025;17(13): 2153. DOI: 10.3390/nu17132153. PMID: 40647258.

23. Staley C, Weingarden AR, Khoruts A, Sadowsky MJ. Interaction of gut microbiota with bile acid metabolism and its influence on disease states. Appl Microbiol Biotechnol. 2017;101(1):47-64. DOI: 10.1007/s00253-016-8006-6. PMID: 27888332.

24. Gerbe F, Legraverend C, Jay P. The intestinal epithelium tuft cells: specification and function. Cell Mol Life Sci. 2012;69(17):2907-17. DOI: 10.1007/s00018-012-0984-7. PMID: 22527717.

25. Qiu P, Ishimoto T, Fu L, Zhang J, Zhang Z, Liu Y. The Gut Microbiota in Inflammatory Bowel Disease. Front Cell Infect Microbiol. 2022;12:733992. DOI: 10.3389/fcimb.2022.733992. PMID: 35273921.

26. Kai Y, Intestinal villus structure contributes to even shedding of epithelial cells. Biophys. J. 2021;120:699-710. DOI: 10.1016/j.bpj.2021.01.003. PMID: 33453270.

27. Pelaseyed T, Bergström JH, Gustafsson JK, Ermund A, Birchenough GM, Schütte A, et al. The mucus and mucins of the goblet cells and enterocytes provide the first defense line of the gastrointestinal tract and interact with the immune system. Immunol Rev. 2014;260(1):8-20. DOI: 10.1111/imr.12182. PMID: 24942678.

28. Postler TS, Ghosh S. Understanding the Holobiont: How Microbial Metabolites Affect Human Health and Shape the Immune System. Cell Metab. 2017;26(1):110-30. DOI: 10.1016/j.cmet.2017.05.008. PMID: 28625867.

29. Desai MS, Seekatz AM, Koropatkin NM, Kamada N, Hickey CA, Wolter M, et al. A Dietary Fiber-Deprived Gut Microbiota Degrades the Colonic Mucus Barrier and Enhances Pathogen Susceptibility. Cell. 2016;167(5):1339-53.e21. DOI: 10.1016/j.cell.2016.10.043. PMID: 27863247.

30. Mu Q, Kirby J, Reilly CM, Luo XM. Leaky Gut as a Danger Signal for Autoimmune Diseases. Front Immunol. 2017;8:598. DOI: 10.3389/fimmu.2017.00598. PMID: 28588585.

31. Okada T, Fukuda S, Hase K, Nishiumi S, Izumi Y, Yoshida M, et al. Microbiota-derived lactate accelerates colon epithelial cell turnover in starvation-refed mice. Nat Commun. 2013;4:1654. DOI: 10.1038/ncomms2668. PMID: 23552069.

32. Koh A, De Vadder F, Kovatcheva-Datchary P, Bäckhed F. From Dietary Fiber to Host Physiology: Short-Chain Fatty Acids as Key Bacterial Metabolites. Cell. 2016;165(6):1332-45. DOI: 10.1016/j.cell.2016.05.041. PMID: 27259147.

33. Marchix J, Goddard G, Helmrath MA. Host-Gut Microbiota Crosstalk in Intestinal Adaptation. Cell Mol Gastroenterol Hepatol. 2018;6(2):149-62. DOI: 10.1016/j.jcmgh.2018.01.024. PMID: 30023411.

34. Kayama H, Okumura R, Takeda K. Interaction Between the Microbiota, Epithelia, and Immune Cells in the Intestine. Annu Rev Immunol. 2020;38:23-48. DOI: 10.1146/annurev-immunol-070119-115104. PMID: 32340570.

35. Gill SK, Rossi M, Bajka B, Whelan K. Dietary fibre in gastrointestinal health and disease. Nat Rev Gastroenterol Hepatol. 2021;18(2):101-16. DOI: 10.1038/s41575-020-00375-4. PMID: 33208922.

36. Mathers JC. Dietary fibre and health: the story so far. Proc Nutr Soc. 2023;82(2):120-9. DOI: 10.1017/S0029665123002215. PMID: 36786062.

37. Wang DD, Li Y, Bhupathiraju SN, Rosner BA, Sun Q, Giovannucci EL, et al. Fruit and Vegetable Intake and Mortality: Results From 2 Prospective Cohort Studies of US Men and Women and a Meta-Analysis of 26 Cohort Studies. Circulation. 2021;143(17):1642-54. DOI: 10.1161/CIRCULATIONAHA.120.048996. PMID: 33641343.

38. Okawa Y, A Discussion of Whether Various Lifestyle Changes can Alleviate the Symptoms of Irritable Bowel Syndrome. Healthcare. 2022;10:2011. DOI: 10.3390/healthcare10102011. PMID: 36292457.

39. Meyer A, Carbonnel F, Dahm CC, Halkjær J, Tjønneland A, Schulze MB, et al. Fruits, Vegetables, Legumes, and Potatoes and Risk of Crohn's Disease and Ulcerative Colitis. Am J Gastroenterol. 2026;121(3):733-44. DOI: 10.14309/ajg.0000000000003602. PMID: 40553119.

40. Nagarajan N, Morden A, Bischof D, King EA, Kosztowski M, Wick EC, Stein EM. The role of fiber supplementation in the treatment of irritable bowel syndrome: a systematic review and meta-analysis. Eur J Gastroenterol Hepatol. 2015;27(9):1002-10. DOI: 10.1097/MEG.0000000000000425. PMID: 26148247.

41. Ioniță-Mîndrican CB, Ziani K, Mititelu M, Oprea E, Neacșu SM, Moroșan E, et al. Therapeutic Benefits and Dietary Restrictions of Fiber Intake: A State of the Art Review. Nutrients. 2022;14(13):2641. DOI: 10.3390/nu14132641. PMID: 35807822.

42. Böhn L, Störsrud S, Törnblom H, Bengtsson U, Simrén M. Self-reported food-related gastrointestinal symptoms in IBS are common and associated with more severe symptoms and reduced quality of life. Am J Gastroenterol. 2013;108(5):634-41. DOI: 10.1038/ajg.2013.105. PMID: 23644955.

43. El-Salhy M, Gundersen D. Diet in irritable bowel syndrome. Nutr J. 2015;14:36. DOI: 10.1186/s12937-015-0022-3. PMID: 25880820.

44. Nabi A, Lateef I, Nisa Q, Banoo A, Rasool RS, Shah MD, et al. Phaseolus vulgaris-Colletotrichum lindemuthianum Pathosystem in the Post-Genomic Era: An Update. Curr Microbiol. 2022;79(2):36. DOI: 10.1007/s00284-021-02711-6. PMID: 34982236.

45. Monk JM, Wu W, Lepp D, Pauls KP, Robinson LE, Power KA. Navy Bean Supplementation in Established High-Fat Diet-Induced Obesity Attenuates the Severity of the Obese Inflammatory Phenotype. Nutrients. 2021;13(3):757. DOI: 10.3390/nu13030757. PMID: 33652785.

46. Vieira NM, Peghinelli VV, Monte MG, Costa NA, Pereira AG, Seki MM, et al. Beans comsumption can contribute to the prevention of cardiovascular disease. Clin Nutr ESPEN. 2023;54:73-80. DOI: 10.1016/j.clnesp.2023.01.007. PMID: 36963901.

47. Zhang X, Irajizad E, Hoffman KL, Fahrmann JF, Li F, Seo YD, et al. Modulating a prebiotic food source influences inflammation and immune-regulating gut microbes and metabolites: insights from the BE GONE trial. EBioMedicine. 2023;98:104873. DOI: 10.1016/j.ebiom.2023.104873. PMID: 38040541.

Received: 18 Dec 2025
Accepted: 29 Mar 2026
Published: 31 Mar 2026

Cite in Vancouver style as: Vasylyeva IM, Nakonechna OA, Makieieva NI, Yarmysh NV, Denysenko SA. Diet and the microbiome: Interaction pathways in chronic intestinal diseases (literature review). Inter Collegas. 2026;13(1):8p. In press. https://doi.org/10.35339/ic.2026.13.1.vnm