SIKORA, Jakub, KNYCHALSKA, Karolina, ŁABUDA, Mikołaj, KRÓLIKOWSKA, Klaudia, SŁOJEWSKA, Aleksandra, KOTKOWIAK, Agata, SOWIŃSKA, Teresa, MENTEL, Oliwia, BOGUCKA, Adrianna and SZEMA, Agnieszka. The Impact of Alcohol on the Gut Microbiota: A literature review. Journal of Education, Health and Sport. 2025;80:60045. eISSN 2391-8306.
https://doi.org/10.12775/JEHS.2025.80.60045
https://apcz.umk.pl/JEHS/article/view/60045

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).
Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).
The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 03.04.2025. Revised: 06.05.2025. Accepted: 06.05.2025. Published: 09.05.2025.

The Impact of Alcohol on the Gut Microbiota: A literature review

Authors:

Jakub Sikora

Profi-Med Medical Center Goleniów Marii Konopnickiej 10A

72-100 Goleniów

esiak10play@gmail.com

https://orcid.org/0009-0007-9637-0709

Karolina Knychalska

University Clinical Hospital no. 2 PMU in Szczecin, Powstańców Wielkopolskich 72 St 70-111 Szczecin

karolinaknychalska@gmail.com

https://orcid.org/0009-0003-3736-0579

Mikołaj Łabuda

Independent Public Voivodeship Integrated Hospital in Szczecin, ul. Arkońska 4

71-455 Szczecin

labuda.mikolaj@gmail.com

https://orcid.org/0009-0002-4137-4319

Klaudia Królikowska
University Clinical Hospital no. 2 PMU in Szczecin, Powstańców Wielkopolskich 72 St 70-111 Szczecin
klaudia.1799@wp.pl
https://orcid.org/0009-0007-7984-4642

Aleksandra Słojewska
Independent Public Voivodeship Integrated Hospital in Szczecin, ul. Arkońska 4
71-455 Szczecin
o.slojewska@gmail.com
https://orcid.org/0009-0007-7532-0948

Agata Kotkowiak
Family Medicine Clinic "Podgórna", Podgórna 22 St.
70-205 Szczecin Poland
akotkowiak@gmail.com
https://orcid.org/0009-0004-4797-6980

Teresa Sowińska
University Clinical Hospital no. 2 PMU in Szczecin, Powstańców Wielkopolskich 72 St 70-111 Szczecin
tsowinska@icloud.com
https://orcid.org/0009-0003-0061-212X

Oliwia Mentel
University Clinical Hospital no. 2 PMU in Szczecin, Powstańców Wielkopolskich 72 St 70-111 Szczecin
oliwiamentel@gmail.com
https://orcid.org/0009-0004-4739-0621

Adrianna Bogucka
Independent Public Voivodeship Integrated Hospital in Szczecin, ul. Arkońska 4
71-455 Szczecin
adrianna.bogucka@icloud.com
https://orcid.org/0009-0001-8870-0495

Agnieszka Szema
University Clinical Hospital no. 2 PMU in Szczecin, Powstańców Wielkopolskich 72 St 70-111 Szczecin
aga.szema@gmail.com
https://orcid.org/0009-0000-5017-3426

Abstract

Introduction and Purpose

The gut microbiota plays a key role in maintaining the body's homeostasis, influencing metabolic, immunological, and neurological processes. Alcohol consumption, particularly in excessive amounts, disrupts the composition and function of the intestinal microbiota, leading to dysbiosis. This article explores the mechanisms by which alcohol affects the gut microbiota and its consequences for overall health. Additionally, it discusses potential therapeutic strategies aimed at mitigating these negative effects.

State of Knowledge

Chronic alcohol consumption results in a decrease in the number of beneficial bacteria, such as Lactobacillus and Bifidobacterium, while pathogenic microorganisms increase, which weakens the intestinal barrier and increases inflammatory reactions. Alcohol affects the microbiota through cytotoxic mechanisms, changes in intestinal pH, modulation of the immune system and effects on bacterial metabolism and the gut-brain axis. Alcohol-related gut dysbiosis contributes to the development of a number of conditions, including alcoholic liver disease, metabolic disorders, neurodegenerative diseases and increased cancer risk

Conclusion

Understanding the impact of alcohol on the gut microbiota is essential for preventing and managing its negative health consequences. Therapeutic approaches such as probiotic and

prebiotic supplementation, a fiber-rich diet, and gut microbiota transplantation may help rebalance the gut microbiota and reduce negative effects of alcohol consumption.

Keywords: alcohol; gut microbiota; dysbiosis; probiotics; alcoholic liver disease; gut-brain axis

Introduction

In 2016, alcohol use accounted for 2.2% of deaths in women and 6.8% of deaths in men, and disability-adjusted life years (DALYs) were 2.3% in women and 8.9% in men [1]. Alcohol addiction carries many negative health consequences [2]. Alcohol, being a commonly consumed psychoactive substance, has multiple effects on the human body, affecting the nervous system, metabolism and inflammation [3]. Its negative effects are also observed in the quality of the gut microbiota, as well as its function [4]. The gut microbiota is a complex ecosystem of microorganisms that perform key functions in maintaining the body's homeostasis, regulating metabolic, immune and neurological processes [5], [6]. It consists mainly of bacteria, but also includes archaeons, viruses and fungi [7]. Disorders of its composition can lead to serious health consequences, including chronic inflammation and metabolic diseases [8]. Alcohol consumption, especially in excessive amounts, is one of the important factors contributing to intestinal dysbiosis, which consequently affects the functioning of the entire body [9], [10]. One solution to counteract the effects of alcohol on the gut microbiota may be probiotics, including bifidobacteria and lactobacilli [11].
The purpose of this review is to present the current state of knowledge regarding the effects of alcohol on the gut microbiota and potential therapeutic strategies to modulate it.

Changes in the composition of the intestinal microbiota

Research results indicate that chronic alcohol consumption leads to significant changes in the quantitative and qualitative composition of the intestinal microbiota [12]. When this occurs, there can be a significant reduction in the abundance of bacteria with probiotic properties, such as Lactobacillus and Bifidobacterium, with a concomitant increase in pathogenic

bacteria, including Proteobacteria, Enterobacteriaceae and Escherichia coli [13]. These changes lead to a disruption of intestinal barrier function, resulting in increased permeability of the intestinal epithelium and an increased inflammatory response by the body [14]. Alcohol can affect intestinal bacteria, contributing to dysbiosis and/or bacterial tanslocation [15], [16].Both alcohol abuse and inflammatory bowel diseases (IBD) are characterized by a decrease in the number of Lactobacillus spp. in the gut and an increase in Enterobacteriaceae [17]. Lactobacillus spp. regulate pH, increase the secretion of mucus and antimicrobial peptides [18]. They increase digestive efficiency, improve the integrity of the gastrointestinal barrier, and create competition for opportunistic pathogens [19]. The anti-inflammatory and immunomodulatory functions of Lactobacillus spp. in intestinal inflammation are also known [20]. Bifidobacterium adolescentis is among the species of intestinal microbiota that is described as most closely related to dietary habits [21], [22]. All Bifidobacterium, thanks to such structures as pili or fimbriae, can communicate with host cells through adhesion [23]. Bifidobacterium longum, thanks to its regulation of tryptophan metabolism and production of Indole-3-carbaldehyde (I3C), participates in the interaction of the gut-skin axis, which helps alleviate atopic dermatitis [24]. Reduction of bacteria such as, Lachnospiraceae, Roseburia, Faecalibacterium, and Blautia can contribute to the overgrowth of intestinal bacteria, results in an increase in endotoxins in the blood, with genera such as Klebsiella, and Lactococcus taking their place [25]. A study by Yuan et al. showed that Klebsiella pneumoniae producing high levels of alcohol (HiAlc Kpn) can be associated with up to 60% of people with nonalcoholic fatty liver disease (NAFLD) [26]. Chronic alcohol consumption and the associated increase in Klebsiella pneumoniae bacteria may also contribute to an increased risk for Klebsiella-associated pneumonia [27]. Moreover, when toxic microbial compounds such as ethanol, lipopolysaccharides and endotoxins produced by Proteobacteria, especially Klebsiella pneumoniae, and aflatoxins produced by Aspergillus species are produced in the gut, mitochondrial and peroxisome dysfunction can occur [28].

Studies have shown that alcohol consumption also alters the composition of the fungal microbiota, increasing the abundance of Candida albicans, which can lead to candidiasis and increased toxin production [29]. Long-term dysbiosis can also affect the metabolism of vitamins, especially B and K vitamins, which are synthesized by intestinal bacteria [30].
The synthesis of vitamins by intestinal bacteria is not coincidental, as there are differences between them, such as the fact that dietary-derived B vitamins are absorbed to the greatest extent in the small intestine, while most bacterial B vitamins are produced and absorbed in the

large intestine [31]. It is the B vitamins in the distal part of the large intestine that can act as nutrients for the host and its microbiota, regulators of immune cell activity, mediators of drug efficacy, factors that promote the survival or fitness of certain bacteria, and as suppressors of colonization by pathogenic bacteria and modulators of colonic inflammation [32].
Vitamin B12 deficiency, which occurs as a result of intestinal dysbiosis, has the potential to contribute to stroke pathogenesis, severity, and treatment outcomes [33].
Moreover, thyroid function is closely linked to nutrition through the diet-gut-thyroid axis, as micronutrients such as iodine, selenium, iron, zinc, copper, magnesium, vitamin A and vitamin B12 affect the synthesis and regulation of thyroid hormones throughout life [34].

Mechanisms of alcohol's effects on the microbiota

Alcohol affects the intestinal microbiota through a number of mechanisms, such as cytotoxic effects, alteration of gastrointestinal pH, modulation of the immune response, damage to the intestinal barrier, and effects on the gut-brain axis [35], [36]. Alcohol increases the expression of pro-inflammatory proteins such as TNF-α and IL-6, which promotes the loosening of tight junctions and bacterial translocation. Increased permeability of the intestinal barrier allows pathogens to penetrate, which can lead to infection and exacerbate inflammation [37]. Gram negative bacteria produce lipopolysaccharide, which are among the more potent inducers of Toll-like receptor(TLR) proteins [38]. Chronic consumption of alcoholic beverages exacerbates the permeability of the intestinal barrier, which promotes the penetration of products of the intestinal microbiota into the lymph and portal circulation [39]. In such a mechanism, pathogen-associated molecular patterns (PAMPs) enter the liver, where cells of the reticuloendothelial system are stimulated, these in turn stimulate monocytes [40]. This process results in the production of cytokines and chemokines characteristic of the inflammatory response [41]. TLR4 is activated by PAMPs, resulting in the induction of NF--κB and the deletion of the chemokine CC(CCL2) and IL-8, due to the activation of this cascade, macrophages and neutrophil granulocytes penetrate the liver structure [42]. Alcohol also increases the expression of pro-inflammatory proteins such as TNF-α and IL-6, their increased levels can be observed in acute alcoholic hepatitis(AH) and affect the worse prognosis of patients [39]. When sterile inflammation is observed, alcohol affects cell apoptosis and loss of stability [43]. The main mechanism, occurring in acute alcoholic hepatitis, in which alcohol affects cell death is activation of the mitochondrial-dependent

apoptotic pathway and dysfunction of the endoplasmic reticulum [44].

Alcohol consumption can increase intestinal permeability by damaging the intestinal mucosa. This causes harmful and potentially toxic endotoxins to enter the systemic circulation, contributing to alcoholic liver disease [45].

Changes in the gut microbiota increase acetaldehyde production through bacterial metabolism of ethanol [46].

Health consequences

Alcoholic liver disease (ALD)

The liver, due to its location, is in constant contact with metabolites and gut-derived bacterial cultures, so disturbances in the composition of the microbiota affect the development of liver diseaseb [47]. Intestinal dysbiosis contributes to the development and progression of hepatic steatosis, inflammation and cirrhosis through bacteremia, increased serum lipopolysaccharide levels and increased bacterial translocation due to increased intestinal permeability [48]. In chronic alcoholism, changes in the microbiota can lead to liver and other organ damage, with Bacteroidetes and Proteobacteria being the main contributors [49]. In liver cirrhosis associated with alcohol dependence, the commensal microbiota is depleted [50]. In addition, there is an increase in the bacterial culture of Proebacteria and Fusobacteria, with a concomitant decrease in Bacteroidetes [51].

Metabolic diseases

Gut dysbiosis can lead to metabolic disorders, including insulin resistance, metabolic syndrome and type 2 diabetes [52]. Changes in the gut microbiota can also affect lipid metabolism and the production of hormones, such as leptin, that regulate appetite and energy metabolism [53], [54]. In a study by Machiro Otaka et al. it was proven that serum leptin levels in rats significantly decrease after alcohol intake. Therefore, it can be presumed that a significant effect on metabolic diseases such as obesity is due to alcohol intake [55].

Neurological disorders

Disorders of the gut microbiota affect the gut-brain axis, which can contribute to the development of depression, anxiety and neurodegenerative diseases. Changes in the gut microbiota can also affect neuroinflammatory processes that are associated with the development of diseases such as Alzheimer's [56], and Parkinson's [57].

In Alzheimer's disease, increased permeability of the gut and blood-brain barrier caused by dysbiosis of the microbiota may influence the pathogenesis of AD and other neurodegenerative disorders, especially those associated with aging [58].

Moreover, knowledge of the alteration of the gut microbiota and its effect on changes in brain activity opens up therapeutic possibilities, taking into account work on the microbiome of a person with a neurological disorder [59].

Studies show this relationship through the formation of bacterial amyloids. Lipopolysaccharides and bacterial amyloids synthesized by the intestinal microbiota can w activate an immune response leading to neuroinflammation [60].

Metabolites of the gut microbiota and its effects on host neurochemical changes may also increase or decrease AD risk, such as GABA, 5-HT, BMAA, vitamin biosynthesis and NMDA receptor and BDNF expression [61].

Increased risk of cancer

Alcohol is a type I carcinogen, and according to the WHO, it caused 5% of all deaths in 2016, 13% of which were due to cancer. Among gastrointestinal cancers, the association is clear for esophageal, liver and colorectal cancers, and more debatable for stomach and pancreatic cancers [62]. Alcohol induces chronic inflammation in the gut and liver, which can promote cancerous processes, particularly in the gastrointestinal tract [63]. Intestinal microbes can convert ethanol and its metabolites, including the toxic acetaldehyde, which can induce the development of colorectal cancer [64]. Colorectal cancer (CRC) carcinogenic mechanisms associated with the intestinal microbiota involve imbalances in the intestinal microbiota. They also include invasion and colonization of pathogenic microorganisms and impaired intestinal barrier function induced by microbial metabolites and virulence factors [65]. There are two models (“alpha-bug” and “driver-passenger”) of intestinal microbial carcinogenesis related to CRC [66].

Therapeutic strategies

Several therapeutic strategies are being considered to minimize the negative effects of alcohol

on the gut microbiota:

The use of probiotics containing Lactobacillus and Bifidobacterium strains has been shown to have beneficial effects on restoring microbiota balance and reducing inflammation. Some probiotics merely pass through, while others permanently colonize the intestines, allowing for increased efficiency of metabolic activity [67]. One therapeutic option in patients with alcohol abuse disorders is to transplant fecal microbiota from a healthy donor, which could improve microbiota diversity, as well as the production of short-chain fatty acids (SCFAs) [68]. Personalized treatment, which would be based on the composition of patients' microbiota and the selection of an individual donor for fecal matter transplantation, might seem more promising [69].

Summary

Alcohol abuse, as well as its occasional consumption, can affect the state of the gut microbiota. First of all, it leads to intestinal dysbiosis, which is associated with further negative health consequences. Alcohol consumption contributes to a decrease in the abundance of such bacteria as, Lactobacillus and Bifidobacterium. At the same time, the abundance of Proteobacteria, Enterobacteriaceae and Escherichia increases. Through these changes, pro-inflammatory factors are activated while positive immunomodulatory mechanisms are reduced. An altered gut microbiota then also means greater growth of fungi, particularly Candida albicans. It is important to pay attention to the effects of alcohol consumption on the gut microbiota in order to prevent them early enough, or to seek solutions to reverse these negative effects. Decreased production of vitamins by intestinal bacteria, as well as disturbances in the composition and quantity of micronutrients can be the beginning of more serious disorders. Increased serum lipopolysaccharide levels and increased bacterial translocation due to increased intestinal permeability can lead to alcoholic liver disease and the subsequent progression of hepatic steatosis. Evidence of neuroinflammatory processes that take place in intestinal dysbiosis should also not escape attention. Cancers that are related to alcohol abuse have also been widely reported. In colorectal cancer, the gut microbiota plays one of the key roles. Various therapeutic strategies are being developed using probiotics, as well as transplantation of fecal matter from donors. Further research should focus on therapeutic processes that could reduce the negative effects of alcohol on the gut microbiota to reduce disease progression.

Disclosure

Author's contribution:

Conceptualization: Jakub Sikora, Agata Kotkowiak, Oliwia Mentel

Methodology: Teresa Sowińska, Klaudia Królikowska, Jakub Sikora

Formal analysis: Aleksandra Słojewska, Karolina Knychalska,

Investigation: Aleksandra Słojewska, Teresa Sowińska, Oliwia Mentel

Data curation: Klaudia Królikowska, Agata Kotkowiak, Adrianna Bogucka

Writing: Jakub Sikora, Mikołaj Łabuda, Agnieszka Szema

Supervision: Teresa Sowińska, Karolina Knychalska, Adrianna Bogucka

Project administration: Mikołaj Łabuda, Agnieszka Szema, Klaudia Królikowska

Receiving funding: Not applicable.

All authors have read and agreed with the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Not applicable.

Acknowledgements: Not applicable.

Conflicts of Interest: The authors declare no conflict of interest.

Declaration of the use of generative AI and AI-assisted technologies in the writing process.

In preparing this work, the authors used ChatGPT for the purpose of improving language and readability. After using this tool, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

References

[1] S. C. Wang, Y. C. Chen, S. J. Chen, C. H. Lee, and C. M. Cheng, "Alcohol Addiction, Gut Microbiota, and Alcoholism Treatment: A Review," Int J Mol Sci, vol. 21, no. 17, pp. 1–11, Sep. 2020, doi: 10.3390/IJMS21176413.

[2] A. F. Carvalho, M. Heilig, A. Perez, C. Probst, and J. Rehm, "Alcohol use disorders," *The Lancet*, vol. 394, no. 10200, pp. 781–792, Aug. 2019, doi: 10.1016/S0140-6736(19)31775-1

[3] M. Butts, V. L. Sundaram, U. Murughiyan, A. Borthakur, and S. Singh, "The Influence of Alcohol Consumption on Intestinal Nutrient Absorption: A Comprehensive Review," *Nutrients*, vol. 15, no. 7, Apr. 2023, doi: 10.3390/NU15071571.

[4] C. H. Kuo, L. L. Wu, H. P. Chen, J. Yu, and C. Y. Wu, "Direct effects of alcohol on gut-epithelial barrier: Unraveling the disruption of physical and chemical barrier of the gut-epithelial barrier that compromises the host-microbiota interface upon alcohol exposure," *J Gastroenterol Hepatol*, vol. 39, no. 7, pp. 1247–1255, Jul. 2024, doi: 10.1111/JGH.16539.

[5] Y. Zang, X. Lai, C. Li, D. Ding, Y. Wang, and Y. Zhu, "The Role of Gut Microbiota in Various Neurological and Psychiatric Disorders-An Evidence Mapping Based on Quantified Evidence," *Mediators Inflamm*, vol. 2023, 2023, doi: 10.1155/2023/5127157.

[6] W. M. De Vos, H. Tilg, M. Van Hul, and P. D. Cani, "Gut microbiome and health: mechanistic insights," *Gut*, vol. 71, no. 5, pp. 1020–1032, May 2022, doi: 10.1136/GUTJNL-2021-326789.

[7] J. Wang, N. Zhu, X. Su, Y. Gao, and R. Yang, "Gut-Microbiota-Derived Metabolites Maintain Gut and Systemic Immune Homeostasis," *Cells*, vol. 12, no. 5, Mar. 2023, doi: 10.3390/CELLS12050793.

[8] E. Thursby and N. Juge, “Introduction to the human gut microbiota,” *Biochem J*, vol. 474, no. 11, pp. 1823–1836, Jun. 2017, doi: 10.1042/BCJ20160510.

[9] K. Pohl, P. Moodley, and A. D. Dhanda, “Alcohol’s Impact on the Gut and Liver,” *Nutrients*, vol. 13, no. 9, Sep. 2021, doi: 10.3390/NU13093170.

[10] V. B. Dubinkina *et al.*, “Links of gut microbiota composition with alcohol dependence syndrome and alcoholic liver disease,” *Microbiome*, vol. 5, no. 1, 2017, doi: 10.1186/S40168-017-0359-2.

[11] S. Leclercq, P. Stärkel, N. M. Delzenne, and P. de Timary, “The gut microbiota: A new target in the management of alcohol dependence?,” *Alcohol*, vol. 74, pp. 105–111, Feb. 2019, doi: 10.1016/J.ALCOHOL.2018.03.005.

[12] A. W. Yan *et al.*, “Enteric dysbiosis associated with a mouse model of alcoholic liver disease,” *Hepatology*, vol. 53, no. 1, pp. 96–105, Jan. 2011, doi: 10.1002/HEP.24018.

[13] P. A. Engen, S. J. Green, R. M. Voigt, C. B. Forsyth, and A. Keshavarzian, “The Gastrointestinal Microbiome: Alcohol Effects on the Composition of Intestinal Microbiota,” *Alcohol Res*, vol. 37, no. 2, p. 223, Jun. 2015, Accessed: Mar. 31, 2025. [Online]. Available: https://pmc.ncbi.nlm.nih.gov/articles/PMC4590619/

[14] C. H. Kuo, L. L. Wu, H. P. Chen, J. Yu, and C. Y. Wu, “Direct effects of alcohol on gut-epithelial barrier: Unraveling the disruption of physical and chemical barrier of the gut-epithelial barrier that compromises the host–microbiota interface upon alcohol exposure,” *J Gastroenterol Hepatol*, vol. 39, no. 7, pp. 1247–1255, Jul. 2024, doi: 10.1111/JGH.16539.

[15] Z. Xu *et al.*, “Chronic alcohol exposure induced gut microbiota dysbiosis and its correlations with neuropsychic behaviors and brain BDNF/Gabra1 changes in mice,” *Biofactors*, vol. 45, no. 2, pp. 187–199, Mar. 2019, doi: 10.1002/BIOF.1469.

[16] R. Yue, X. Wei, J. Zhao, Z. Zhou, and W. Zhong, “Essential Role of IFN-γ in Regulating Gut Antimicrobial Peptides and Microbiota to Protect Against Alcohol-Induced Bacterial Translocation and Hepatic Inflammation in Mice,” *Front Physiol*, vol. 11, Jan. 2021, doi: 10.3389/FPHYS.2020.629141.

[17] P. V. Kuprys *et al.*, “Alcohol decreases intestinal ratio of Lactobacillus to Enterobacteriaceae and induces hepatic immune tolerance in a murine model of DSS-colitis,” *Gut Microbes*, vol. 12, no. 1, pp. 1–16, Nov. 2020, doi: 10.1080/19490976.2020.1838236.

[18] S. Rastogi and A. Singh, “Gut microbiome and human health: Exploring how the probiotic genus Lactobacillus modulate immune responses,” *Front Pharmacol*, vol. 13, p. 1042189, Oct. 2022, doi: 10.3389/FPHAR.2022.1042189/PDF.

[19] E. Dempsey and S. C. Corr, “Lactobacillus spp. for Gastrointestinal Health: Current and Future Perspectives,” *Front Immunol*, vol. 13, Apr. 2022, doi: 10.3389/FIMMU.2022.840245.

[20] F. Cristofori, V. N. Dargenio, C. Dargenio, V. L. Miniello, M. Barone, and R. Francavilla, “Anti-Inflammatory and Immunomodulatory Effects of Probiotics in Gut Inflammation: A Door to the Body,” *Front Immunol*, vol. 12, Feb. 2021, doi: 10.3389/FIMMU.2021.578386.

[21] M. Satti, M. Modesto, A. Endo, T. Kawashima, P. Mattarelli, and M. Arita, “Host-Diet Effect on the Metabolism of Bifidobacterium,” *Genes (Basel)*, vol. 12, no. 4, Apr. 2021, doi: 10.3390/GENES12040609.

[22] L. A. Bolte *et al.*, “Long-term dietary patterns are associated with pro-inflammatory and anti-inflammatory features of the gut microbiome,” *Gut*, vol. 70, no. 7, pp. 1287–1298, Jul. 2021, doi: 10.1136/GUTJNL-2020-322670.

[23] M. O’Connell Motherway *et al.*, “A Bifidobacterial pilus-associated protein promotes colonic epithelial proliferation,” *Mol Microbiol*, vol. 111, no. 1, pp. 287–301, Jan. 2019, doi: 10.1111/MMI.14155.

[24] Z. Fang *et al.*, “Bifidobacterium longum mediated tryptophan metabolism to improve atopic dermatitis via the gut-skin axis,” *Gut Microbes*, vol. 14, no. 1, 2022, doi: 10.1080/19490976.2022.2044723.

[25] V. B. Dubinkina *et al.*, “Links of gut microbiota composition with alcohol dependence syndrome and alcoholic liver disease,” *Microbiome*, vol. 5, no. 1, pp. 1–14, Oct. 2017, doi: 10.1186/S40168-017-0359-2/FIGURES/4.

[26] J. Yuan *et al.*, “Fatty Liver Disease Caused by High-Alcohol-Producing Klebsiella pneumoniae,” *Cell Metab*, vol. 30, no. 4, pp. 675-688.e7, Oct. 2019, doi: 10.1016/J.CMET.2019.08.018/ATTACHMENT/8F608090-C959-4DCF-8C63-AB4D23DF0597/MMC4.PDF.

[27] D. R. Samuelson *et al.*, “Alcohol-associated intestinal dysbiosis impairs pulmonary host defense against Klebsiella pneumoniae,” *PLoS Pathog*, vol. 13, no. 6, p. e1006426, 2017, doi: 10.1371/JOURNAL.PPAT.1006426.

[28] T. P. T. Pham, M. T. Alou, M. H. Golden, M. Million, and D. Raoult, “Difference between kwashiorkor and marasmus: Comparative meta-analysis of pathogenic characteristics and implications for treatment,” *Microb Pathog*, vol. 150, Jan. 2021, doi: 10.1016/J.MICPATH.2020.104702.

[29] H. Chu *et al.*, “The Candida albicans exotoxin candidalysin promotes alcohol-associated liver disease,” *J Hepatol*, vol. 72, no. 3, pp. 391–400, Mar. 2020, doi: 10.1016/J.JHEP.2019.09.029.

[30] L. Voland, T. Le Roy, J. Debédat, and K. Clément, “Gut microbiota and vitamin status in persons with obesity: A key interplay,” *Obes Rev*, vol. 23, no. 2, Feb. 2022, doi: 10.1111/OBR.13377.

[31] C. T. Peterson, D. A. Rodionov, S. N. Peterson, and A. L. Osterman, “B Vitamins and Their Role in Immune Regulation and Cancer,” *Nutrients 2020, Vol. 12, Page 3380*, vol. 12, no. 11, p. 3380, Nov. 2020, doi: 10.3390/NU12113380.

[32] T. Uebanso, T. Shimohata, K. Mawatari, and A. Takahashi, “Functional Roles of B-Vitamins in the Gut and Gut Microbiome,” *Mol Nutr Food Res*, vol. 64, no. 18, Sep. 2020, doi: 10.1002/MNFR.202000426.

[33] W. Roth and M. Mohamadzadeh, “Vitamin B12 and gut-brain homeostasis in the pathophysiology of ischemic stroke,” *EBioMedicine*, vol. 73, Nov. 2021, doi: 10.1016/J.EBIOM.2021.103676.

[34] L. Fan *et al.*, “Gut microbiota bridges dietary nutrients and host immunity,” *Sci China Life Sci*, vol. 66, no. 11, pp. 2466–2514, Nov. 2023, doi: 10.1007/S11427-023-2346-1.

[35] N. Qamar, D. Castano, C. Patt, T. Chu, J. Cottrell, and S. L. Chang, “Meta-analysis of alcohol induced gut dysbiosis and the resulting behavioral impact,” *Behavioural Brain Research*, vol. 376, Dec. 2019, doi: 10.1016/j.bbr.2019.112196.

[36] F. Bishehsari *et al.*, “Alcohol and Gut-Derived Inflammation,” *Alcohol Res*, vol. 38, no. 2, p. 163, 2017, Accessed: Mar. 31, 2025. [Online]. Available: https://pmc.ncbi.nlm.nih.gov/articles/PMC5513683/

[37] E. J. Pone, “Analysis by Flow Cytometry of B-Cell Activation and Antibody Responses Induced by Toll-Like Receptors,” *Methods Mol Biol*, vol. 1390, pp. 229–248, Jan. 2016, doi: 10.1007/978-1-4939-3335-8_15.

[38] R. F. Schwabe, E. Seki, and D. A. Brenner, “Toll-like receptor signaling in the liver,” *Gastroenterology*, vol. 130, no. 6, pp. 1886–1900, 2006, doi: 10.1053/J.GASTRO.2006.01.038.

[39] E. J. Pone, “Analysis by Flow Cytometry of B-Cell Activation and Antibody Responses Induced by Toll-Like Receptors,” *Methods Mol Biol*, vol. 1390, pp. 229–248, Jan. 2016, doi: 10.1007/978-1-4939-3335-8_15.

[40] E. Qi *et al.*, “Uncovering the impact of alcohol on internal organs and reproductive health: Exploring TLR4/NF-kB and CYP2E1/ROS/Nrf2 pathways,” *Animal Model Exp Med*, vol. 7, no. 4, pp. 444–459, Aug. 2024, doi: 10.1002/AME2.12436.

[41] F. Ezquer *et al.*, “Innate gut microbiota predisposes to high alcohol consumption,” *Addiction biology*, vol. 26, no. 4, Jul. 2021, doi: 10.1111/ADB.13018.

[42] S. Leclercq, C. De Saeger, N. Delzenne, P. De Timary, and P. Stärkel, “Role of inflammatory pathways, blood mononuclear cells, and gut-derived bacterial products in alcohol dependence,” *Biol Psychiatry*, vol. 76, no. 9, pp. 725–733, 2014, doi: 10.1016/J.BIOPSYCH.2014.02.003.

[43] R. G. Thurman, “II. Alcoholic liver injury involves activation of Kupffer cells by endotoxin,” *Am J Physiol*, vol. 275, no. 4, 1998, doi: 10.1152/AJPGI.1998.275.4.G605.

[44] Y. S. Roh, B. Zhang, R. Loomba, and E. Seki, “TLR2 and TLR9 contribute to alcohol-mediated liver injury through induction of CXCL1 and neutrophil infiltration,” *Am J Physiol Gastrointest Liver Physiol*, vol. 309, no. 1, Jul. 2015, doi: 10.1152/AJPGI.00031.2015.

[45] J. A. Eom *et al.*, “Gut-microbiota prompt activation of natural killer cell on alcoholic liver disease,” *Gut Microbes*, vol. 15, no. 2, Dec. 2023, doi: 10.1080/19490976.2023.2281014.

[46] J. Person, “Alcohol and the small intestine,” *Scand J Gastroenterol*, vol. 26, no. 1, pp. 3–15, 1991, doi: 10.3109/00365529108996478.

[47] C. Martino *et al.*, “Acetate reprograms gut microbiota during alcohol consumption,” *Nature Communications 2022 13:1*, vol. 13, no. 1, pp. 1–11, Aug. 2022, doi: 10.1038/s41467-022-31973-2.

[48] N. Y. Lee and K. T. Suk, “The Role of the Gut Microbiome in Liver Cirrhosis Treatment,” *International Journal of Molecular Sciences 2021, Vol. 22, Page 199*, vol. 22, no. 1, p. 199, Dec. 2020, doi: 10.3390/IJMS22010199.

[49] H. Tilg, P. D. Cani, and E. A. Mayer, “Gut microbiome and liver diseases,” *Gut*, vol. 65, no. 12, pp. 2035–2044, Dec. 2016, doi: 10.1136/GUTJNL-2016-312729.

[50] V. B. Dubinkina *et al.*, “Links of gut microbiota composition with alcohol dependence syndrome and alcoholic liver disease,” *Microbiome*, vol. 5, no. 1, pp. 1–14, Oct. 2017, doi: 10.1186/S40168-017-0359-2/FIGURES/4.

[51] Y. Chen *et al.*, “Characterization of fecal microbial communities in patients with liver cirrhosis,” *Hepatology*, vol. 54, no. 2, pp. 562–572, Aug. 2011, doi: 10.1002/HEP.24423.

[52] C. O. Iatcu, A. Steen, and M. Covasa, “Gut Microbiota and Complications of Type-2 Diabetes,” *Nutrients 2022, Vol. 14, Page 166*, vol. 14, no. 1, p. 166, Dec. 2021, doi: 10.3390/NU14010166.

[53] M. A. Pelleymounter *et al.*, “Effects of the obese gene product on body weight regulation in ob/ob mice,” *Science*, vol. 269, no. 5223, pp. 540–543, 1995, doi: 10.1126/SCIENCE.7624776.

[54] M. W. Schwartz, R. J. Seeley, L. A. Campfield, P. Burn, and D. G. Baskin, “Identification of targets of leptin action in rat hypothalamus,” *J Clin Invest*, vol. 98, no. 5, pp. 1101–1106, Sep. 1996, doi: 10.1172/JCI118891.

[55] M. Otaka *et al.*, “Effect of alcohol consumption on leptin level in serum, adipose tissue, and gastric mucosa,” *Dig Dis Sci*, vol. 52, no. 11, pp. 3066–3069, Nov. 2007, doi: 10.1007/S10620-006-9635-X.

[56] C. Chen *et al.*, “Gut microbiota regulate Alzheimer’s disease pathologies and cognitive disorders via PUFA-associated neuroinflammation,” *Gut*, vol. 71, no. 11, pp. 2233–2252, 2022, doi: 10.1136/GUTJNL-2021-326269.

[57] M. F. Sun and Y. Q. Shen, “Dysbiosis of gut microbiota and microbial metabolites in Parkinson’s Disease,” *Ageing Res Rev*, vol. 45, pp. 53–61, Aug. 2018, doi: 10.1016/J.ARR.2018.04.004.

[58] C. Jiang, G. Li, P. Huang, Z. Liu, and B. Zhao, “The Gut Microbiota and Alzheimer’s Disease,” *J Alzheimers Dis*, vol. 58, no. 1, pp. 1–15, 2017, doi: 10.3233/JAD-161141.

[59] F. Angelucci, K. Cechova, J. Amlerova, and J. Hort, “Antibiotics, gut microbiota, and Alzheimer’s disease,” *J Neuroinflammation*, vol. 16, no. 1, May 2019, doi: 10.1186/S12974-019-1494-4.

[60] A. Megur, D. Baltriukienė, V. Bukelskienė, and A. Burokas, “The Microbiota-Gut-Brain Axis and Alzheimer’s Disease: Neuroinflammation Is to Blame?,” *Nutrients*, vol. 13, no. 1, pp. 1–24, Jan. 2020, doi: 10.3390/NU13010037.

[61] X. Hu, T. Wang, and F. Jin, “Alzheimer’s disease and gut microbiota,” *Science China Life Sciences 2016 59:10*, vol. 59, no. 10, pp. 1006–1023, Aug. 2016, doi: 10.1007/S11427-016-5083-9.

[62] G. Vanella, L. Archibugi, S. Stigliano, and G. Capurso, “Alcohol and gastrointestinal cancers,” *Curr Opin Gastroenterol*, vol. 35, no. 2, pp. 107–113, Mar. 2019, doi: 10.1097/MOG.0000000000000502.

[63] S. Grad, L. Abenavoli, and D. L. Dumitrascu, “The Effect of Alcohol on Gastrointestinal Motility,” *Rev Recent Clin Trials*, vol. 11, no. 3, pp. 191–195, Aug. 2016, doi: 10.2174/1574887111666160815103251.

[64] V. B. Dubinkina *et al.*, “Links of gut microbiota composition with alcohol dependence syndrome and alcoholic liver disease,” *Microbiome*, vol. 5, no. 1, pp. 1–14, Oct. 2017, doi: 10.1186/S40168-017-0359-2/FIGURES/4.

[65] Z. Wang, W. Dan, N. Zhang, J. Fang, and Y. Yang, “Colorectal cancer and gut microbiota studies in China,” *Gut Microbes*, vol. 15, no. 1, 2023, doi: 10.1080/19490976.2023.2236364.

[66] Y.-N. Yu and J.-Y. Fang, “Gut Microbiota and Colorectal Cancer,” *Gastrointest Tumors*, vol. 2, no. 1, pp. 26–32, 2015, doi: 10.1159/000380892.

[67] Y. Xiao, J. Zhao, H. Zhang, Q. Zhai, and W. Chen, “Mining Lactobacillus and Bifidobacterium for organisms with long-term gut colonization potential,” *Clinical Nutrition*, vol. 39, no. 5, pp. 1315–1323, May 2020, doi: 10.1016/J.CLNU.2019.05.014/ASSET/3E93A56F-9544-40CB-9402-F3A28FD11F12/MAIN.ASSETS/GR3.SML.

[68] K. Pohl, P. Moodley, and A. D. Dhanda, “Alcohol’s Impact on the Gut and Liver,” *Nutrients 2021, Vol. 13, Page 3170*, vol. 13, no. 9, p. 3170, Sep. 2021, doi: 10.3390/NU13093170.

[69] V. B. Dubinkina *et al.*, “Links of gut microbiota composition with alcohol dependence syndrome and alcoholic liver disease,” *Microbiome*, vol. 5, no. 1, pp. 1–14, Oct. 2017, doi: 10.1186/S40168-017-0359-2/FIGURES/4.