Al-Qadisiyah J. Vet. Med. Sci. 2025; 24 (3): 10.29079/qjvms.2025.166156.1127
The 1st National and International Scientific Conference November 26 / 2025
ISSN P: 1818-5746
E: 2313-4429
qjvms@qu.edu.iq

Diet–Microbiome–Immune Crosstalk in Mice

Hussein I. Kareem [1] Ali Hassan Daghir [2]
Department of Veterinary Microbiology, College of Veterinary Medicine, University of Al-Qadisiyah, Iraq

Submitted: October 13, 2025
Revised: November 18, 2025
Accepted: November 19, 2025

Correspondence:
Ali Hassan Daghir
ali.janabi@qu.edu.iq

Abstract Recent mouse and translational studies are connecting nutrients to barrier control and immune tone. High fat diets increase permeability, endotoxemia, and inflammation. Fiber and prebiotics enrich immune tolerant, short-chain-fatty-acid producers. Bile acids act as microbes and host signals. Polyphenols restructure communities and lower pro-inflammatory signals. Purified diets reduce variability and increase reproducibility. Modern analytics delineate the diet, metabolite, and immune response triad. They include metagenomics, metatranscriptomics, targeted metabolomics, and flow cytometry. It suggests employing precise diets and tracking microbial metabolites over time. Immune responses should be evaluated with specific assays. The intestinal barrier must be protected and reinforced. High fiber and polyphenol diets are preferable. For causal questions, gnotobiotic and humanized models are the most dependable. Combined, multi-omics tools and immune phenotyping should be used. These increase strength and reproducibility of studies on diet, microbiome, and immune associations. They assist in the translational progression of laboratory data towards both preventive and therapeutic applications.

Keywords: bile acids; diet; gut microbiome; immunity; short-chain fatty acids

Introduction Among the most rapidly modifiable determinants of gut microbiota composition is diet. Restricting even a single macronutrient—be it fat, fiber, protein, or a plant-derived phytochemical—alters the microbial community structure, and its subsequent metabolic byproducts, including some which are short-chain fatty acids. SCFAs are the primary macromolecular metabolic byproducts of anaerobic digestion and SCFAs serve as the main energy source for colonocytes in addition to promoting the closure of the tight junctions (1). Bile acids modulate SCFA levels, and, through FXR and TGR5, SCFA immune antagonism. Following gut microbiome dysbiosis, a phenomenon commonly referred to as intestinal permeabilities the condition known as ("leaky gut syndrome") and subsequent elevations in systemic inflammation. These impacts link dietary patterns to targeted and systemic physiology—inclusive of metabolic function, infection susceptibility, and the gut–brain–immune axis. Research in 2020–2025 supports this in both mice and humans (2-4). Advancements in science have been made in various fields. For example, the accuracy of long-read sequencing in identifying strains has improved. Furthermore, metabolic and metagenomics techniques have been developed to associate microbes to their metabolic products. Flow cytometry and cytokine analysis have demonstrated the immunological changes that occur. There is also the use of controlled experiments that maintain a dietary intervention using defined, fully supplemented macronutrient and micronutrient balances. Eating patterns have been aligned to the circadian rhythms of the organisms. Some study designs include the administration of probiotics or polyphenols when the research question permits. These designs permit analysis of the clear causal chain illustrated: Diet → Microbe → Metabolite → Receptor → Immune Output. This review follows ten main topics from the outline and provides guidance for sound study design and result interpretation (5–10).

Overview of the Gut Microbiome

The gut of a mouse is a habitat for thick and varied populations of microbes. Their composition is largely influenced by dietary intake. Consumption of a high-fat diet is associated with reduced diversity of gut microbes

Al-Qadisiyah J. Vet. Med. Sci. 2025; 24 (3): 10.29079/qjvms.2025.166156.1127
The 1st National and International Scientific Conference November 26 / 2025
ISSN P: 1818-5746
E: 2313-4429
qjvms@qu.edu.iq

and a higher concentration of pathogenic, endotoxin-producing bacteria. A fiber-rich diet positively correlates with an increase in butyrate-producing microbes and strengthens gut barrier tolerance. These shifts in microbial populations associate with increased fat mass, insulin resistance, low-grade systemic inflammation, and heightened anxiety-like behavior. These outcomes have been demonstrated in multiple studies (11-15). The microbial world's tiny products are of utmost importance. Butyrate serves as an energy source for colon cells, strengthens cell junctions, and stimulates T cell and Treg proliferation via GPCR and HDAC inhibition. Propionate as well as acetate have metabolic and immunological actions that extend beyond the gut. Bile acids convey microbial signals to immune cells via FXR, TGR5, and VDR. Products of tryptophan enhance epithelial defenses via AhR. Together, these pathways start to illuminate the connections between nutrition, microbes, and immune system states (16–20).

The Immune System and Its Relationship with the Microbiome

The immune system learns from the gut microbiome. SCFAs also have the capacity to expand Tregs as well as modulate the activity of different subsets of T helper cells to control inflammation. TGF-β, IL-10 and IL-5 promote IgA secretion. Bile acid receptors modulate the activity of dendritic cells and macrophages which in turn shapes immune responses. Together these inputs help control the balance of immune tolerance and inflammation at the mucosal surfaces and beyond (3, 4, 11, 16, 20). Circuits governed by the immune system are impacted by diet. An increased consumption of fats is associated with increased concentrations of blood lipopolysaccharides (LPS), increased barrier weakness, and increased levels of TNF-α and IL-6. The cytokine and prebiotic levels are suppressed, while the barrier polyphenols and certain probiotics strengthen the junctions and restore the beneficial genera Akkermansia and Bifidobacterium. The resultant changes shift immunity towards tolerance and better the metabolic profiles (21-23). Signals extend towards additional organs. Microbial signals and airway immunity are linked by the gut-lung axis. The SCFAs and tryptophan metabolites connect diet, microbes, and the mental state through the gut-brain axis. Systemic and bile-acid signaling affects the liver as well as peripheral metabolism. The microbiome and Vitamin D status interact, which could affect anti-cancer immune mechanisms. These connections illustrate the non-digestive health impacts diet can have (4, 9).

Microbial metabolism and Immune modulation

Microbial communities in the gut convert food into dietary bioactive components. The main classes are SCFAs, bile acids, and tryptophan derivatives. They each process through different receptors and result in distinct immune outcomes. SCFAs enhance the epithelial barriers and the anti-inflammatory cascades. Bile acids transmit metabolic and immune communication between the gut and the liver. Tryptophan metabolites modulate cytokine imbalance and defend the mucosa. These metabolites function as integrative nodes of intricate pathways merging nutrition, immunity, and sustained homeostasis. SCFAs, in particular, are agonists to receptors, GPR41, GPR43, and GPR109A and inhibit HDACs. These routes expand Tregs. They curb pro-inflammatory cytokines. They improve barrier protein expression. They also alter neutrophil and macrophage function. Butyrate has the most potent effect locally. Propionate and acetate have the most potent systemic effects. These three converge to adjust epithelial and immune cell secretion towards tolerance (3, 16, 19). Bile acids function as signaling molecules. Microbes deconjugate and dehydroxylate primary bile acids. The resulting pool engages FXR and TGR5 on epithelial cells and immune cells. These receptors send signals that regulate antimicrobial peptides, inflammasome activity, and cytokine release. Furthermore, they influence the maturation of dendritic cells and the polarization of macrophages. Disrupted bile-acid pools are associated with intestinal inflammation and metabolic disease. These pathways—FXR and TGR5—can be targeted as possible means to restore balance. The diet provides the substrates for these conversions. The type and amount of fat shifts bile flow and microbial enzymes (4, 11, 17, 20). Tryptophan metabolites function through the aryl hydrocarbon receptor (AhR). Indoles synthesized by microbes aid in the strengthening of the gut barrier. They also provide instructive signals to innate lymphoid cells. AhR activation has anti-inflammatory properties and promotes the repair of epithelial surfaces. Resolving stress with sufficient dietary tryptophan also fortifies the gut lining. The gut microbiota is also balanced alongside restoration of SCFA concentrations. This indicates that diet has direct control over the microbiome–immune system interface (9). All metabolite interactions are very intricate and diverse. In the liver, SCFAs can affect the synthesis of bile acids. Bile acids, in turn, can modify bacteria populations, creating feedback systems. Some of these systems involve polyamine and amino acid derivatives. Certain polyphenols are dual acting "duplibiotics." They stimulate beneficial bacteria while inhibiting pathogenic bacteria. The lower oligomers also engage the host receptors. Under high fat conditions, anthocyanins reduce gut permeability and inflammation and increase Akkermansia and Bifidobacterium and restore SCFAs (18, 23).

Microbiota-Driven Shaping of Systemic Immunity

Al-Qadisiyah J. Vet. Med. Sci. 2025; 24 (3): 10.29079/qjvms.2025.166156.1127
The 1st National and International Scientific Conference November 26 / 2025
ISSN P: 1818-5746
E: 2313-4429
qjvms@qu.edu.iq

Events in the gut have ramifications for the rest of the body. Metabolites of microbiota move across the gut and enter the systemic circulation and lymphatic system. These penetrate the liver, adipose tissue, lungs, and even the brain. SCFAs can attenuate inflammation and insulin sensitiveness, and even extend Tregs beyond the gut. Bile-acid signals control liver metabolism and the immune system. These signals also alter the activities of monocytes and macrophages. All these signals have an impact on the biome since diet determines the function of the microbiota (20). Disruptions from a high-fat diet are myriad. They increase gut permeability and plasma levels of LPS and promote persistently low-grade inflammation. Mice increase the production of TNF-α and IL-6, modify tight-junction proteins scatter the intestinal barrier. Some exhibit anxiety-like behaviors and glial alterations in the brain. A high-fat diet, in particular, is post-antibiotic therapy, is shown to persistently alter the metabolism of sorbitol intolerance. This occurs due to the elimination of Clostridia, a genus of bacteria responsible for the hydrolysis of sorbitol. These interactions indicate an intricate relationship between diet, microbes, and the host. (15).Microbial metagenome has protective patterns. Fiber fermentation and prebiotic oligosaccharides support keystone species that rebuild SCFA pools, reduce pro-inflammatory cytokines, and lower epithelial permeability. Polyphenols give similar support in high-fat models. They restore barrier integrity and improve tissue redox balance. Certain probiotics, for instance, Lactobacillus acidophilus, actively promote lower levels of lipid accumulation and insulin resistance, in addition to inflammation. This shifts systemic immunity balance toward positive change and improves metabolic health (21–23).Microbial signals are important to many diseases. In the case of cancer, it is now the diet–microbiota–immunity aspects that are integrated into the care plan for the microbiome. Understanding how to promote SCFA-producing microbes and lower inflammation through diet improves the response to therapy on a plant-based and Mediterranean diet. The gut–brain–metabolic axis connects dietary patterns to mood and cognition. There are reviews that link these pathways to Parkinson's disease and other conditions such as insulin resistance and cognitive decline. These are all links that require further study, but fit well into the current mechanisms that have been observed in animal work (24–26).Any good experimental design requires the measurement of systemic outcomes that have been captured as a whole. Measures of the liver, adipose tissue, lung, and as relevant, the brain, should all be captured. Serum metabolomics and proteomics should be applied. Consider immune profiling from the blood and spleen. Sample collection should be physiologically matched with feeding and circumferential rhythm. When studying the effects of stress on the brain, include models of aggression. These steps illustrate the way gut alterations influence bodywide immunity and support clinical application (7).

Mouse Strains and Genetic Backgrounds

There is considerable variation even among different strains of mice which has effects on their immune tone, microbiota, and sensitivity to diets. Under the influences of a high fat diet, the mice differ in their cytokine and metabolic activity, and fiber and polyphenol responsiveness. These effects are further modified by vendor, housing, and pathogen status, which are important variables to record, as they help explain the variation observed in different labs. The beneficial effects of germ-free mice is that they can test complex hypotheses by direct cause-effect methodology in the framework of defined, colonizable interactions. They also can be gnotobiotic, which consists of synthetic models that contain constructed bacterial communities and humanized mice that are colonized by human microbiota. The former are designed to study the external mouse microbiota, while the latter emphasizes dietary reactions of humanized mice. Collectively, these models help in bridging the gaps between the host and microbes that can modify the outcomes being studied. These complex systems are very powerful, yet, economically costly, thus they should be used judiciously within focused mechanistic frameworks.Bile-acid and tryptophan pathways are influenced by genetics, as are the bile-acid and microbial metabolism pathways. Host enzymes and transporters interact with microbial metabolites. Host Vitamin D receptor pathways interact with and extend this bile-acid microbial metabolism cancer immunity nexus in some reports. These host pathways chronicle the variation in immune outcomes in different strains on the same diet and also demonstrate therapeutic targets (4, 11, 20, 24).

Analytical approaches

Taxonomic profiling is a good starting point. For a rapid assessment of diversity and community composition, use 16S rRNA. For community structure, shotgun metagenomics provides better resolution to the species and strain levels. For optimal assemblies, combine the depth of Illumina with long reads. State versions of the databases used in the analyses. Upload the code and parameter sets used to perform the operations. When feasible, the target of a few validation checks should be qPCR. These steps increase trust on the results obtained (25–26, 28–30).

Function mapping is the next step. Use metagenomics to estimate the abundance of specific enzyme families involved in the degradation of carbohydrates, the

Al-Qadisiyah J. Vet. Med. Sci. 2025; 24 (3): 10.29079/qjvms.2025.166156.1127
The 1st National and International Scientific Conference November 26 / 2025
ISSN P: 1818-5746
E: 2313-4429
qjvms@qu.edu.iq

conversion of bile acids, and the production of SCFAs. Integrate metatranscriptomics to visualize the instructor of active pathways. Use targeted metabolomics for the production of SCFAs, bile acids, tryptophan, and other downstream metabolites. Relate the concentration of these metabolites to their corresponding gene fragments and transcript levels. This connects nutrition, microbiomes, and derivates. It connects to a host's receptors and immune outputs. "Host immune system measures" Use flow cytometry to measure Treggs, Th subsets, innate lymphoid cells and macrophages and dendritic cells. Run a panel of cytokines to measure concentration of TNF-α, IL-6, IL-10, etc. Immunostaining or western blotting can be used to measure concentration of specific proteins such as tight junction proteins. Measure cell permeability using FITC-dextran. Add histology to measure the degree of inflammation and damage to the epithelium. Use proteomics to measure protein concentration when there are expected systemic signals. Together, these layers illustrate the mechanism and the effects. "Host immune system measures" statistical plans should be made. Outcomes should be specified in advance. Multivariate techniques should be used to analyze multiple taxa at the same time and make connections across all layers of omics. To ensure the reliability of the study, associations should be validated in independent cohorts or across time points. The data, along with the processed data, should be shared to the public. This supports the reuse and comparison of the research and speeds up peer analysis as well as meta analysis.

High fat diets and chronic inflammation

High fat diets are with a lack of dietary fiber, are with a lack of dietary fiber, are with a lack of dietary fiber. They increase the ability to cross both the unaware and the side junctions. They elevate the concentration of LPS in the plasma. There is a gain of chronic inflammation. Studies show an increase of IL and TNF-α concentration in the body when feeding a high fat diet. The concentration of tight junction proteins decreases. New openings in the duodenum appear. Some models show altered mind coordination with brain glial cells and the enteric system. Following these changes, behavior is modified. These effects can be observed and do not always means there is an increase in weight." They can occur as early as begining of a person's life. They can also continue throughout a person's life (1–2, 6–8, 15).In the case of high-fat consumption the bile-acid pools are altered. The transformations conducted by microbes shift with the continuous changes. The changes with the bile-acid pools FXR and TGR5 receptor signaling alters epithelial barriers and immune system homeostasis. Liver metabolism also gets altered. This physiology integrates the gut's activity with physiology at whole body level. Activating bile-acid receptors in some studies have been shown to reduce inflammation and improve metabolism. The strongest influence in metabolism comes from the diet as the primary driver that determines bile flow and the substrates supplied to the system (4, 11, 17, 20). The high-fat diet, in combination with other stressors, poses a challenge. The use of antibiotics in the past has been shown to trigger the phenomenon of 'long-lasting intolerances'. One study reported remnants of a sorbitol intolerance from a previous episode of abuse with combined antibiotics and high-fat diet exposure. This occurred due to the loss of the Clostridia that usually breaks down the sorbitol. Environmental toxins of dysbiosis, like the methylmercury, are amplified with the addition of high-fat conditions. These conclusions illustrate that diet never functions in isolation; the total of exposures must be taken into account (2, 27).Protective strategies are effective. SCFA levels have been shown to equilibrate via fibers and prebiotics. The supplements also close gaps in the epithelial barriers and reduce inflammation. Polyphenols deepen the epithelial oxidative balance and improve epithelial defense. Some probiotics like Lactobacillus acidophilus have been shown to reduce fat gain and inflammation in high fat-diet mice. All these strategies together shift the immune state towards tolerance and also better systemic metabolism. The use of combined strategies in clinical practice often leads to greater benefits. Controlled experimental designs help to assess the real effect size (18, 21–23).

Supplementation of High Fibers and Prebiotics

Dietary Fibers serve as energy sources for beneficial microbes. They improve the Putative Butyrate-Producing Microbiota and increase SCFA levels. Butyrate is a major energy source for colonocytes, It also fortifies tight junctions and diminishes inflammation. Supplementary to the systemic benefits of propionate and acetate, diets rich in fiber in both mice and humans are associated with anti-inflammatory beneficial microbiota and improved metabolic profiles. These outcomes are pervasive in the literature (1–3, 16, 24–26).Prebiotics show greater target specificity. The compounds inulin, fructooligosaccharides (FOS), and galactooligosaccharides (GOS) also promote more butyrate-producing bacteria. They also demonstrate the ability to decrease gut leakiness and gut cytokine levels in high-fat-fed models. The FOS and GOS study conducted in 2021 corroborates the ability of FOS and GOS to counteract dysbiosis caused by high-fat consumption. They also demonstrated anti-inflammatory properties and bone structural preservation. Additional studies also confirm the improvement of tight-junction

Al-Qadisiyah J. Vet. Med. Sci. 2025; 24 (3): 10.29079/qjvms.2025.166156.1127
The 1st National and International Scientific Conference November 26 / 2025
ISSN P: 1818-5746
E: 2313-4429
qjvms@qu.edu.iq

proteins and metabolic health upon the consumption of prebiotics. This evidence solidifies the understanding that consumption of fiber and prebiotic foods are effective nutritional strategies to restore proper balance within the gut and immune system. These data reinforce the suggestion that prebiotics are barrier-protective agents (22). Polyphenols act in tandem with fiber. Anthocyanins reconstruct beneficial taxa and enhance barrier function through the attenuation of inflammatory mediators in high-fat settings and restoration of redox balance. These attributes are beneficial in the restoration of tolerance to the gut. The combination of polyphenols with prebiotics is likely to yield greater benefits than using either ingredient alone. This is likely due to the action of the prebiotics in enhancing production of SCFAs, remodeling bile acids, and improving the antioxidant effect (18, 23).

Diet-Microbiome Studies (Metagenomics)

Relating diets to microbial activities is the focus of metagenomics. It calculates the carbohydrate-utilizing genes and toxins the organism has, bile-acid conversion, and SCFA production, and assesses stress-response pathways. Within metagenomics, microbial communities in high-fiber conditions demonstrate a polysaccharide digestive locus. It reveals the presence of genes in the pathway of butyrate production. In high-fat conditions, it reveals an increased proportion of bile-acid modification genes. It may also reveal factors of virulence and the pathways of endotoxins. These patterns coincide with outcomes for barriers and immunity (18, 25-26, 28-30).The depth of shotgun is important. Gene quantification is aided by the breadth of coverage provided by Illumina reads. Better contig assembly and operon context are provided by long reads. Strain-level resolution is provided by hybrid methods of sequencing. They also enhance the discovery of low-abundance players. These details are vital when the effects arise from a small number of sequences. It is crucial to convey the precise versions of the reporting databases and version parameters employed. Confidence is raised by the validation from targeted qPCR (25-26, 28-30). The power of metagenomics is enhanced by the addition of metabolomics. SCFAs, bile acids, and tryptophan metabolites must be analyzed and measured. Correlate the metabolite concentrations with gene and transcript levels. Employ linkage models to associate the genes with the resulting products and the host receptors. Use metatranscriptomics to determine which genes are expressed for particular diets. This elucidates mechanism. It also pinpoints the targets of intervention.For instance, absence of bile salt hydrolase genes may point to potential probiotics or bile-acid modulators (18, 23, 25-25-26).

Conclusion

The diet influences the microflora of the intestine, which produce metabolites that communicate with the immune system. Barr.ier integrity is key but also weakened by fat while strengthened by fiber and polyphenols

Acknowledgement

This authors would like to thank the College of Veterinary Medicine, University of Al-Qadisiyah, Iraq.

Funding

Self-funded by the author.

Conflict of Interest

The author declares no conflict of interest

References

1. Blaak EE, Canfora EE, Theis S, Frost G, Groen AK, Mithieux G, et al. Short chain fatty acids in human gut and metabolic health. Benef Microbes. 2020;11(2):135-155. doi:10.3920/BM2019.0057. https://doi.org/10.3920/BM2019.0057
2. Tyagi A, Yadav S, Nirmal J, Verma R, Singh R. The gut microbiota-bile acid axis: a crucial regulator of immune responses. Front Immunol. 2025;16:1510593. https://doi.org/10.1007/s11274-025-04395-7
3. Zhang L, Wang Y, Liu Z, Xu J, Ni H. Protein modification by short-chain fatty acids in sepsis. Front Immunol. 2023;14:1286245. https://doi.org/10.3389/fimmu.2023.1171834
4. Wang Y, Tong Q, Shou D, Ma L, Wen S, Liu W, et al. Insights into the bile acid-gut microbiota axis in intestinal failure-associated liver disease. Aliment Pharmacol Ther. 2022;55(5):575-594. https://doi.org/10.1111/apt.16676
5. Liaqat I, Ahsan H, Rubab S, Zahra N, Afzal A, Qadri I, et al. Modified diet and humanized microbiota induce metabolic endotoxemia in mice. Biomed Pharmacother. 2021;139:111658.
6. Mindikoglu AL, Abdulsada MM, Jain A, Choi J, Jalal PK, Devaraj S, et al. Intermittent fasting from dawn to sunset for 30 days induces protective serum proteome changes. J Proteomics. 2020;217:103645. https://doi.org/10.1016/j.jprot.2020.103645
7. Hosseinkhani F, Heinken A, Thiele I, Lindenburg PW, Harms AC, Hankemeier T. Gut bacterial metabolites in immune signaling and NCDs. Nutrients. 2021;13(8):2733. PubMed https://doi.org/10.1080/19490976.2021.1882927
8. Ashaolu TJ. Immune-boosting functional foods and mechanisms. Microbiol Res. 2020;239:126494.
9. Guan B, Huang G, Dong S, Li J, Tian J, He Y, et al. Bile acid coordinates microbiota homeostasis and

Al-Qadisiyah J. Vet. Med. Sci. 2025; 24 (3): 10.29079/qjvms.2025.166156.1127
The 1st National and International Scientific Conference November 26 / 2025
ISSN P: 1818-5746
E: 2313-4429
qjvms@qu.edu.iq

systemic immunometabolism. Signal Transduct Target Ther. 2022;7:206.
10. Luu M, Monning H, Visekruna A. SCFAs and food allergy mechanisms. Allergy. 2020;75(6):1336-1348.
11. Sun R, Yang N, Kong B, Cao B, Feng D, Yu X. Critical roles of bile acids in regulating intestinal mucosal immunity. Theranostics. 2021;11(14):6761-6773. https://doi.org/10.1177/17562848211018098
12. Mullish BH, Williams HRT. Bile acid metabolism and C. difficile infection. Ther Adv Gastroenterol. 2021;14:17562848211017725. https://doi.org/10.1177/17562848211017725
13. Nakanishi T, Tamaki Y, Hasegawa T, Hira T, Hara H. High-fat diet alters small-intestinal microbiome and barrier. Nutrients. 2021;13(11):4124.
14. Yao Y, Cai X, Fei W, Ye Y, Zhao M, Zheng C. The role of short-chain fatty acids in immunity and inflammation. Front Immunol. 2022;13:928713. https://doi.org/10.1080/10408398.2020.1854675
15. Boscaini S, Cabrera-Rubio R, Golubeva AV, Moloney RD, Dinan TG, Cryan JF. Microbiota depletion alters whey-protein effects on permeability in HFD mice. Physiol Rep. 2021;9(11):e14867. https://doi.org/10.14814/phy2.14867
16. Losol P, Kim JH. Crosstalk between microbial metabolites and host immunity. Front Immunol. 2024;15:1327438.
17. Thibaut MM, Faucher-Giguère M, Fahmi H, Alessi DR, Bensalma S. Bile-acid-activated receptors and immunity. Cells. 2022;11(3):459.
18. Kang Y, Cai Y, Zhang X, Kong X, Su J, Zhang Y, et al. Lactobacillus acidophilus ameliorates obesity and inflammation in HFD mice. Gut Microbes. 2022;14(1):2041495.
19. Abildinova GZ, Zhumanova G, Mashanbekov AT. Gut-brain-metabolic axis in insulin resistance and cognition. Nutrients. 2024;16(10):1474.
20. Patel D, Das R, Ilango K, Nivetha A, Joseph J. Dietary fatty acids, gut microbiota, and immunity. Clin Nutr ESPEN. 2023;54:1-10.
21. Shi Z, Yuan C, Zhang Y, Huang J, Yan Z, Zhang X, et al. Nuciferine improves HFD-induced obesity and barrier function. J Agric Food Chem. 2021;69(21):5864-5876.
22. Zhang Z, Lin T, Meng Y, Hu M, Shu L, Jiang H, et al. FOS/GOS reverse HFD dysbiosis, permeability, and inflammation. Metabolism. 2021;119:154767. https://doi.org/10.1016/j.metabol.2021.154767
23. Liu D, Liu J, Wang C, et al. Purple sweet potato anthocyanin rebuilds microbiota and barrier in HFD mice. J Food Sci. 2022;87(8):3675-3691. https://doi.org/10.1111/1750-3841.16130
24. Aziz T, Li C, Yu X, et al. Mediterranean diet and gut microbiota: bidirectional links. Nutrients. 2023;15(14):3149.
25. Ciernikova S, Mego M. Diet-driven microbiome changes and physical activity. Nutrients. 2023;15(23):5014. https://doi.org/10.3389/fnut.2023.1285516
26. Mindikoglu AL, Abdulsada MM, et al. Intermittent fasting proteomics and immunity. J Proteomics. 2020;217:103645 (duplicate topic entry consolidated with #6) https://doi.org/10.1016/j.jprot.2020.103645
27. Lee JY, Chen L, Chen H, et al. High fat sustains sorbitol intolerance after antibiotics by loss of Clostridia. Cell Host Microbe. 2024;32(4):612-626.e5.
28. Puljiz Z, Karadža-Lapić L, Perić M, Turk Wensveen T, Potočnjak I. Obesity, gut microbiota, and metabolome. Int J Mol Sci. 2023;24(11):9413.
29. Reznikov EA, Nowak JK, Jasion-Bugajska M, et al. Nutritional therapies in IBD. Nutrients. 2023;15(4):1023. https://doi.org/10.3390/nu15030668
30. Matos GA, Guimarães NK, Pereira RR, et al. Methylmercury and HFD impair barrier and microbiota. Food Chem Toxicol. 2025;192:114281.