Proceedings of ICBioMed 2025 Symposium: AI for Healthcare: Advanced Medical Data Analytics and Smart Rehabilitation
DOI: 10.54254/2753-8818/2025.AU28675

Research Advances on the Impact of Environmental Pollutants on Gut Microbiota

Haibo Wang

Tianjin University of Science and Technology, Tianjin, China
22206106@mail.tust.edu.cn

Abstract. Environmental pollutants such as microplastics, pesticides, and heavy metals are emerging as critical determinants of gut microbiota composition and function. This review synthesizes current evidence from animal models, human studies, and mechanistic investigations to delineate how these contaminants disrupt gut microecology and compromise host health. Across pollutant classes, common pathogenic features include induction of dysbiosis, characterized by depletion of beneficial commensals and enrichment of pro-inflammatory taxa; impairment of intestinal barrier integrity, facilitating translocation of microbial metabolites into systemic circulation; and activation of innate immune signaling pathways. Notably, all three pollutant categories converge on the LPS/TLR4/NF-κB pathway, driving the release of pro-inflammatory cytokines such as TNF-α and IL-6, thereby promoting chronic inflammation and metabolic dysfunction. While animal studies provide robust mechanistic insights, human evidence remains limited, with few large-scale longitudinal cohorts. Future research should prioritize multi-omics, physiologically relevant models, and microbiota-targeted interventions to clarify causal pathways and mitigate pollutant toxicity.

Keywords: Gut microbiota, environmental pollutants, microplastics, pesticides, heavy metals

1. Introduction

With the advancement of global industrialization and agricultural modernization, human exposure pathways to environmental pollutants have diversified beyond inhalation and dermal contact to include significant ingestion routes. This heightened exposure raises critical concerns, as emerging evidence indicates that pollutants not only directly harm host tissues but also significantly disrupt the gut microbiota—a complex ecosystem vital for host immune regulation and metabolic homeostasis [1]. Notably, Claus et al. have proposed a seminal framework outlining four key mechanisms by which pollutants interact with the gut microbiome to impair host health: direct microbial metabolism, re-metabolization via enterohepatic circulation, induction of dysbiosis, and interference with microbial enzymatic activity [1]. While this framework provides a crucial foundation, a significant knowledge gap remains in systematically synthesizing how specific classes of pollutants, particularly microplastics, pesticides, and heavy metals, induce gut microbial alterations that subsequently trigger downstream inflammatory signaling (e.g., the TLR4/NF-kB pathway) and metabolic dysregulation (e.g., glucose/lipid metabolism interference), thereby driving

DOI: 10.54254/2753-8818/2025.AU28675

the development of chronic inflammatory and metabolic comorbidities. Existing reviews often focus on single pollutants or lack mechanistic depth connecting microbial shifts to these specific host pathologies. Therefore, this review aims to comprehensively synthesize current research on the impact of microplastics, pesticides, and heavy metals on the gut microbiota, with a specific focus on elucidating the mechanisms linking pollutant-induced dysbiosis to host inflammatory responses and metabolic dysfunction. By integrating evidence across these pollutant classes, this work seeks to provide an integrative perspective that informs future research directions and potential microbiome-targeted interventions for mitigating pollutant-associated health risks.

2. Effects of microplastics on gut microbiota

2.1. Gut microbiota disruption in animal models

Microplastics (MPs), particularly polystyrene particles, have emerged as environmental contaminants with significant biological consequences [2]. Animal studies consistently show that MP ingestion alters the composition and diversity of the gut microbiota. Xu et al. have found that both 0.5 μm and 50 μm polystyrene MPs significantly shifted gut microbial communities in mice, with marked reductions in beneficial taxa such as Firmicutes and Alphaproteobacteria [3]. These changes were further exacerbated in mice fed a high-fat diet, where MP exposure led to increased abundance of opportunistic pathogens and decreased levels of short-chain fatty acid (SCFA)-producing bacteria. This compositional shift suggests a destabilization of microbial homeostasis and a predisposition toward metabolic and inflammatory disorders.

Additional murine studies support these findings. Jin et al. demonstrated that oral exposure to MPs compromises gut barrier integrity, increases gut permeability, and disrupts microbial-derived metabolite production, including butyrate and acetate [4]. These disruptions are particularly concerning given the critical role of SCFAs in maintaining epithelial health and immunoregulation. Collectively these animal studies indicate that MPs can reshape microbial communities and functional outputs, especially under conditions of metabolic vulnerability.

2.2. Dysbiosis in high-exposure human populations

Human evidence also links microplastic exposure to gut microbiota disturbances. A pioneering study investigated factory workers in Chengdu, China, who experienced high occupational MP exposure [5]. This study combined 16S rDNA sequencing with infrared laser imaging and found significantly higher fecal concentrations of polyurethane and polyethylene microplastics in the high-exposure group compared to controls. These individuals exhibited characteristic dysbiosis, marked by overrepresentation of potential pathobionts, including Bifidobacterium and Streptococcus, with the reduction of SCFA-producing genera such as Ruminococcus torques, Dorea, and Coprococcus. This compositional imbalance is of clinical concern, as SCFA depletion may impair mucosal immunity and increase the risk of inflammatory diseases [6]. The authors concluded that chronic MP exposure, even at sub-toxic levels, may disrupt microbial equilibrium in humans, leading to a shift toward pro-inflammatory microbiota profiles.

2.3. Inflammation and immune activation mechanisms

Emerging studies reveal that MPs not only alter microbial composition but also trigger inflammatory signaling in the gut. Ke et al. observed significantly elevated fecal MP concentrations in preschool children with inflammatory bowel disease (IBD), suggesting a correlation between MP burden and

DOI: 10.54254/2753-8818/2025.AU28675

intestinal inflammation [7]. In controlled animal experiments, Xu et al. showed that 5 μm polystyrene MPs accumulate in the intestines, induce intestinal barrier dysfunction, promote inflammation in the intestinal mucosal lining, and lead to microbial dysbiosis and metabolic disorders [3]. Mechanistically, MPs are believed to enhance bacterial endotoxin translocation due to epithelial barrier disruption, thereby activating innate immune pathways such as TLR4/NF-kB signaling. This activation leads to increased cytokine production (i.e., IL-6 and TNF-alpha), further aggravating mucosal inflammation and microbial dysbiosis [8]. In parallel, MP-induced SCFA depletion reduces the anti-inflammatory buffering capacity of the microbiome, contributing to a feed-forward cycle of immune activation and microbial imbalance.

Furthermore, recent metagenomic analyses have identified enrichment of genes related to oxidative stress, virulence, and horizontal gene transfer under MP exposure, implying enhanced microbial adaptability under stress [9]. These functional changes may amplify the risk of chronic diseases linked to low-grade inflammation and microbiome dysfunction.

Overall, experimental and epidemiological evidence indicates that microplastic exposure disrupts gut microbial composition, impairs intestinal barrier function, and promotes inflammatory signaling cascades. These alterations may underlie the development of metabolic and immune-related disorders. Given the ubiquitous presence of MPs in food and water nowadays, understanding their microbiome-mediated effect is critical for public health and future research should prioritize longitudinal human studies and mechanistic investigations into microbe-host-plastic interactions.

3. Effects of pesticides on gut microbiota

3.1. Gut microbiota disturbance following pesticide residue exposure in animal models

Pesticides can accumulate in humans and animals through the food chain. Due to the potent antimicrobial properties, certain pesticides adversely affect gut microbiota communities. In rodent models, glyphosate and its commercial formulations (i.e., RangerPro/Bioflow) significantly impair the adaptability of core commensal microbiota in the cecum. This effect is mediated by surfactant-facilitated synergistic toxicity and results in a reduction of dominant phyla like Bacteroidota by up to 60%, contributing to pronounced microbial dysbiosis [10]. Almasri et al. demonstrated in invertebrate models that chronic exposure of honeybees to imidacloprid, glyphosate, or difenoconazole–either individually or in combination–alters the gut microbiome composition [11]. Their experiments showed that at environmentally relevant concentrations (0.1 μg/L), such chronic exposure did not significantly disrupt early colonization of core gut microbiota. However, Almasri et al. found thateven at these low doses, pesticides directly impaired key physiological processes by inhibiting detoxification enzymes (i.e., GST), energy metabolism enzymes (i.e., LDH), and oxidative stress defense enzymes (i.e., G6PDH) [11]. Crucially, their research revealed that the presence of core gut microbiota substantially enhanced honeybees' tolerance to pesticide-induced physiological toxicity, whereas microbiota-depleted individuals exhibited heightened sensitivity and exacerbated physiological disorders under identical low-dose exposure.2.2 Dysbiosis in pesticide human populations.

3.2. Dysbiosis in pesticide human populations

Findings from animal models have prompted investigations into whether similar gut microbiota alterations occur in humans. Population-based studies have identified associations between pesticide exposure and microbial dysregulation. In the Dutch Microbiome Project, involving 7,198

DOI: 10.54254/2753-8818/2025.AU28675

participants, occupational pesticide exposure was assessed using a Job-Exposure Matrix (JEM) calibrated with International Standard Classification of Occupations (ISCO) codes. Exposure to insecticides, fungicides, herbicides, and combined pesticide categories was quantified, and statistically significant correlations ($p < 0.05$, FDR-adjusted) were observed between occupational pesticide exposure and altered gut microbial configurations [12]. Environmental exposure studies using Geographic Information System mapping have further demonstrated that residential proximity to agricultural application zones where organophosphate pesticides are applied is associated with shifts in the relative abundance of multiple bacterial taxa and functional alterations in gut metabolic pathways (i.e., methanogenesis from acetate and superpathway of pyridoxal 5'-phosphate biosynthesis and salvage). These findings suggest that chronic low-level organophosphate exposure may contribute to long-term microbial dysregulation in humans [13].

3.3. Mechanistic insights: pesticide-induced gut inflammation

Mechanistic evidence indicates that many pesticides, particularly organophosphates and organochlorines, induce the production of reactive oxygen species (ROS) in the host, leading to oxidative stress [14]. This oxidative imbalance disrupts intestinal epithelial cells, compromises the gut barrier integrity, and alters the redox potential in the intestinal lumen–conditions hostile to obligate anaerobic symbiotic bacteria [15]. Pesticide exposure can trigger intestinal inflammatory responses through multiple mechanisms. First, certain pesticides (e.g., organophosphate and organochlorine herbicides) possess intrinsic cytotoxicity, directly damaging intestinal epithelial cells and disrupting the structural integrity of tight junction proteins such as occluding, claudin, and ZO-1, thereby markedly increasing intestinal permeability [16]. Second, by altering microbial composition, pesticides promote the abnormal proliferation of gram-negative bacteria such as Proteobacteria that produce lipopolysaccharide (LPS), while significantly suppressing beneficial anti-inflammatory bacteria including Lactobacillus and Bifidobacterium [17]. Third, pesticides and their metabolites, such as cytochrome P450 metabolites of organochlorine pesticides, can directly act on immune cells in the lamina propria, activating the NLRP3 inflammasome and modulating the aryl hydrocarbon receptor (AhR) signaling to stimulate the release of pro-inflammatory cytokines such as IL-1β, IL-6, and TNF-α [18]. The interplay of these three mechanisms—barrier disruption, dysbiosis-driven endotoxin accumulation, and immune activation—creates a self-perpetuating inflammatory cycle that can culminate in chronic intestinal inflammation.

Evidences presents a coherent picture of how pesticide exposure perturbs gut microbial communities and contributes to intestinal dysfunction. Experimental data highlight pesticide-driven shifts in microbial composition, human studies confirm associations between exposure and altered microbiota profiles, and mechanistic insights reveal plausible biological pathways linking exposure to chronic inflammation. These findings underscore the role of pesticides as environmental modulators of gut health, with potential consequences for both gastrointestinal and systemic disease risk. Given that pesticides represent only one category of environmental contaminants capable of influencing the gut microbiome, it is essential to also consider other toxicants, such as heavy metals, which share some mechanistic overlaps but may exert distinct compositional and functional impacts. The following section examines the effects of heavy metal exposure on gut microbiota and the resulting implications for host health.

DOI: 10.54254/2753-8818/2025.AU28675

4. Effects of heavy metals on gut microbiota

4.1. Gut microbiota disruption by heavy metal in animal models

Heavy metals have emerged as a global environmental concern due to their persistence, bioaccumulation potential, and toxicity even at low doses [14]. Recent studies indicate that exposure to heavy metal contaminants can disrupt host microbial communities and compromise intestinal homeostasis. In rodent models, George et al. found that chronic lead exposure through drinking water or feed induced significant alterations in gut microbial community structure [19]. Compared to the control group, lead-exposed mice showed marked changes in the abundance of specific bacteria, including Akkermansia, Lactobacillus, Burkholderiaceae, and Bacteroides. Additionally, functional genes associated with metal resistance, antibiotic resistance, and metabolic processes were identified in the gut microbiota of lead-exposed mice.

Cadmium, another widespread environmental contaminant, can disrupt the diversity and composition of gut microbiota. Tao et al. employed histopathology, 16S rDNA sequencing, and metagenomic profiling to examine the effects of 35-day cadmium exposure at concentrations ranging from 6 to 48 mg/L in Cricetulus longicaudatus (long-tailed dwarf hamster) [20]. Cadmium exposure significantly reduced beneficial taxa such as Prevotella and Oscillibacter, while markedly increasing the abundance of potentially pathogenic Treponema. Crucially, despite these specific alterations in bacterial taxa, the overall architecture of the gut microbiota remained largely preserved. These findings suggest that cadmium selectively reshapes gut microbiota composition without completely disrupting ecological stability.

4.2. Dysbiosis in high-exposure human populations

Epidemiological studies in heavy metal-contaminated areas provide evidence that environmental exposure also alters gut microbial communities in humans. In lead-zinc mining regions of Guizhou, China, residents experience chronic multi-metal exposure via dietary chains [21]. Analysis revealed significant correlations between high-level heavy metal exposure (especially arsenic, cadmium, lead, and zinc) and pronounced shifts in gut microbiota. These changes manifested as marked differentiation in microbial beta-diversity and restructuring of specific taxa abundances, with increases in butyrate-producing genera such as Roseburia and Bacteroides, alongside reductions in Prevotella_9. From a clinical perspective, these microbial alterations may have important consequences. Expansion of butyrate-producing genera could reflect a compensatory response to maintain gut barrier integrity, as butyrate is a key energy source for colonocytes and an anti-inflammatory metabolite [22]. However, the concurrent reduction in Prevotella—a genus commonly associated with fiber metabolism and mucosal immune regulation—may compromise intestinal immune function and increase the risk of inflammatory bowel disease (IBD) or metabolic syndrome [23]. Crucially, striking gender disparities were observed. Male residents exhibited more pronounced microbial shifts, including elevated alpha-diversity, compared with females. This disparity may cause higher occupational exposure (mining, smelting, and farming) coupled with lifestyle-related factors such as higher smoking and alcohol consumption rates, which may amplify heavy metal intake. These findings provide indirect but compelling evidence that occupational and behavioral factors exacerbate the gut microbiota-disrupting effects of heavy metals.

DOI: 10.54254/2753-8818/2025.AU28675

4.3. The mechanisms problems caused by heavy metals

Multi-omics analysis reveals that heavy metal exposure disrupts multiple critical metabolic pathways mediated by gut microbiota. This includes significant disturbances in vitamin E metabolism, bile acid metabolism, nitrogen metabolism, energy metabolism, oxidative stress responses, and defense/detoxification mechanisms. For instance, lead exposure may impair nitrogen metabolism by alterations in urease accessory protein UreE and changes in metabolites such as urea and nitrite in the urea cycle [24]. Additionally, lead exposure compromises intestinal barrier integrity. Lead can increase gut permeability, facilitating the translocation of inflammatory cytokines, immune factors, and microbial metabolites into systemic circulation. At the same time, reductions in short-chain fatty acids (SCFAs) caused by lead-induced microbial shifts have been linked to neurological outcomes, including multiple sclerosis-like lesions in animal models [25].

In summary, evidence from both animal models and human studies indicates that heavy metal exposure disrupts gut microbial structure and function, impairs the intestinal barrier, and alters metabolite-mediated host signaling. Mechanistic insights further suggest that these microbial and metabolic disturbances may contribute to systemic pathologies, including inflammatory and neurological disorders. However, compared to the relatively abundant animal data, high-quality human studies remain scarce. Future research should prioritize large-scale, long-term prospective cohort studies to better define the causal links between heavy metal exposure, gut microbiota dysbiosis, and human disease.

5. Conclusion

This review examines the impacts of three major environmental pollutants—microplastics, pesticides, and heavy metals—on the structure and function of the gut microbiota, highlighting the mechanisms by which they perturb gut microecology and compromise intestinal barrier integrity, thereby inducing host metabolic disorders and immune dysfunction. Across pollutant classes, three convergent pathogenic processes emerge: (1) induction of gut microbial dysbiosis, characterized by loss of beneficial commensals and enrichment of pathogenic or pro-inflammatory taxa; (2) impairment of intestinal epithelial barrier integrity, resulting in increased permeability and systemic translocation of microbial-derived metabolites; and (3) activation of key innate immune signaling pathways—particularly the TLR4/NF-κB axis—driving pro-inflammatory cytokine release and metabolic disruption. This unified framework underscores the central role of the gut microbiota in mediating chronic health risks associated with environmental exposures.

However, current evidence remains limited. Mechanistic insights are largely derived from animal models, and extrapolating these findings to complex human systems requires cautious validation. Critically, high-quality, large-scale longitudinal cohort studies are lacking, and exposure assessments remain inadequate in both scope and precision. For instance, while murine studies reveal MP-induced shifts in Firmicutes and Bacteroidetes ratios, human data from occupational cohorts (e.g., Chengdu factory workers) suggest differential dysbiosis patterns. Future research should prioritize physiologically relevant systems (e.g., organoids, multi-omics platforms) to elucidate mechanistic pathways and develop microbiota-targeted strategies for mitigating pollutant toxicity. Furthermore, implementing personalized microbiome monitoring for high-exposure populations—such as occupationally exposed groups or residents in heavily polluted areas—holds promise for early detection of ecological imbalances. By tracking temporal dynamics in microbial α/β diversity, critical functional taxa, and metabolite profiles, this approach could provide early warnings at initial stages of dysbiosis, offering an empirical foundation for developing targeted interventions against

DOI: 10.54254/2753-8818/2025.AU28675

environmental pollutants. Addressing these gaps is crucial for precise environmental health risk assessment and the development of effective preventive measures.

References

[1] Claus, S. P., Guillou, H., & Ellero-Simatos, S. (2016) The gut microbiota: a major player in the toxicity of environmental pollutants? NPJ Biofilms Microbiomes, 2: 16003. https: //doi.org/10.1038/npjbiofilms.2016.3

[2] Yu, H., Zhang, Y., Tan, W., & Zhang, Z. (2022) Microplastics as an Emerging Environmental Pollutant in Agricultural Soils: Effects on Ecosystems and Human Health. Frontiers in Environmental Science, 10. https: //doi.org/10.3389/fenvs.2022.855292

[3] Xu, M., Niu, H., Wu, L., Xing, M., Mo, Z., Chen, Z., Li, X., & Lou, X. (2024) Impact of Microplastic Exposure on Blood Glucose Levels and Gut Microbiota: Differential Effects under Normal or High-Fat Diet Conditions. Metabolites, 14(9). https: //doi.org/10.3390/metabo14090504

[4] Jin, Y., Lu, L., Tu, W., Luo, T., & Fu, Z. (2019) Impacts of polystyrene microplastic on the gut barrier, microbiota and metabolism of mice. Science of The Total Environment, 649: 308-317.

[5] Zhang, X., Wang, H., Peng, S., Kang, J., Xie, Z., Tang, R., Xing, Y., He, Y., Yuan, H., Xie, C., & Liu, Y. (2022) Effect of microplastics on nasal and intestinal microbiota of the high-exposure population. Frontiers in Public Health, 10. https: //doi.org/10.3389/fpubh.2022.1005535

[6] Kim, C. H. (2021) Control of lymphocyte functions by gut microbiota-derived short-chain fatty acids. Cell Mol Immunol, 18(5): 1161-1171. https: //doi.org/10.1038/s41423-020-00625-0

[7] Ke, D., Zheng, J., Liu, X., Xu, X., Zhao, L., Gu, Y., Yang, R., Liu, S., Yang, S., Du, J., Chen, B., He, G., & Dong, R. (2023) Occurrence of microplastics and disturbance of gut microbiota: a pilot study of preschool children in Xiamen, China. EBioMedicine, 97: 104828. https: //doi.org/10.1016/j.ebiom.2023.104828

[8] Cheng, Y., Yang, Y., Bai, L., & Cui, J. (2024) Microplastics: an often-overlooked issue in the transition from chronic inflammation to cancer. J Transl Med, 22(1): 959. https: //doi.org/10.1186/s12967-024-05731-5

[9] Maione, A., Norcia, M., Sinoca, M., Galdiero, M., Maselli, V., Feola, A., Carotenuto, R., Cuomo, P., Capparelli, R., Guida, M., & Galdiero, E. (2023) Polystyrene Microplastics Exacerbate Candida albicans Infection Ability In Vitro and In Vivo. Int J Mol Sci, 25(1). https: //doi.org/10.3390/ijms25010012

[10] Mesnage, R., Panzacchi, S., Bourne, E., Mein, C. A., Perry, M. J., Hu, J., Chen, J., Mandrioli, D., Belpoggi, F., & Antoniou, M. N. (2022) Glyphosate and its formulations Roundup Bioflow and RangerPro alter bacterial and fungal community composition in the rat caecum microbiome. Front Microbiol, 13: 888853. https: //doi.org/10.3389/fmicb.2022.888853

[11] Almasri, H., Liberti, J., Brunet, J. L., Engel, P., & Belzunces, L. P. (2022) Mild chronic exposure to pesticides alters physiological markers of honey bee health without perturbing the core gut microbiota. Sci Rep, 12(1): 4281. https: //doi.org/10.1038/s41598-022-08009-2

[12] Gois, M. F. B., Fernandez-Pato, A., Huss, A., Gacesa, R., Wijmenga, C., Weersma, R. K., Fu, J., Vermeulen, R. C. H., Zhernakova, A., Lenters, V. C., & Kurilshikov, A. (2023) Impact of occupational pesticide exposure on the human gut microbiome. Front Microbiol, 14: 1223120. https: //doi.org/10.3389/fmicb.2023.1223120

[13] Zhang, K., Paul, K., Jacobs, J. P., Cockburn, M. G., Bronstein, J. M., Del Rosario, I., & Ritz, B. (2024) Ambient long-term exposure to organophosphorus pesticides and the human gut microbiome: an observational study. Environ Health, 23(1): 41. https: //doi.org/10.1186/s12940-024-01078-y

[14] Ali, H., Khan, E., & Ilahi, I. (2019) Environmental Chemistry and Ecotoxicology of Hazardous Heavy Metals: Environmental Persistence, Toxicity, and Bioaccumulation. Journal of Chemistry, 2019: 1-14. https: //doi.org/10.1155/2019/6730305

[15] Sharma, T., Sirpu Natesh, N., Pothuraju, R., Batra, S. K., & Rachagani, S. (2023). Gut microbiota: a non-target victim of pesticide-induced toxicity. Gut Microbes, 15(1), 2187578. https: //doi.org/10.1080/19490976.2023.2187578

[16] Liang, Y., Zhan, J., Liu, D., Luo, M., Han, J., Liu, X., Liu, C., Cheng, Z., Zhou, Z., & Wang, P. (2019) Organophosphorus pesticide chlorpyrifos intake promotes obesity and insulin resistance through impacting gut and gut microbiota. Microbiome, 7(1): 19. https: //doi.org/10.1186/s40168-019-0635-4

[17] Liu, W., Li, C., Li, B., Shang, Q., Han, Z., Zhang, Y., Liu, X., Fan, H., Zhang, J., Chen, Y., & Zhang, H. (2022) Lactiplantibacillus plantarum P9 improved gut microbial metabolites and alleviated inflammatory response in pesticide exposure cohorts. iScience, 25(7): 104472. https: //doi.org/10.1016/j.isci.2022.104472

[18] Lima, C., Falcao, M. A. P., Rosa, J. G. S., Disner, G. R., & Lopes-Ferreira, M. (2022) Pesticides and Their Impairing Effects on Epithelial Barrier Integrity, Dysbiosis, Disruption of the AhR Signaling Pathway and

DOI: 10.54254/2753-8818/2025.AU28675

Development of Immune-Mediated Inflammatory Diseases. Int J Mol Sci, 23(20). https: //doi.org/10.3390/ijms232012402

[19] George, S. E., Devereux, R., James, J., Wan, Y., Diamond, G. L., Bradham, K. D., & Thomas, D. J. (2023) Dietary lead modulates the mouse intestinal microbiome: Subacute exposure to lead acetate and lead contaminated soil. Ecotoxicol Environ Saf, 249: 114430. https: //doi.org/10.1016/j.ecoenv.2022.114430

[20] Tao, M., Cao, K., Pu, X., Hou, Y., He, L., Liu, W., Ren, Y., & Yang, X. (2024) Cadmium exposure induces changes in gut microbial composition and metabolic function in long-tailed dwarf hamsters, Cricetulus longicaudatus. Ecol Evol, 14(7): e11682. https: //doi.org/10.1002/ece3.11682

[21] Gao, B., Chi, L., Mahbub, R., Bian, X., Tu, P., Ru, H., & Lu, K. (2017) Multi-Omics Reveals that Lead Exposure Disturbs Gut Microbiome Development, Key Metabolites, and Metabolic Pathways. Chemical Research in Toxicology, 30(4): 996-1005. https: //doi.org/10.1021/acs.chemrestox.6b00401

[22] Geirnaert, A., Calatayud, M., Grootaert, C., Laukens, D., Devriese, S., Smagghe, G., De Vos, M., Boon, N., & Van de Wiele, T. (2017) Butyrate-producing bacteria supplemented in vitro to Crohn's disease patient microbiota increased butyrate production and enhanced intestinal epithelial barrier integrity. Scientific Reports, 7(1). https: //doi.org/10.1038/s41598-017-11734-8

[23] Iljazovic, A., Roy, U., Gálvez, E. J. C., Lesker, T. R., Zhao, B., Gronow, A., Amend, L., Will, S. E., Hofmann, J. D., Pils, M. C., Schmidt-Hohagen, K., Neumann-Schaal, M., & Strowig, T. (2021) Perturbation of the gut microbiome by Prevotella spp. enhances host susceptibility to mucosal inflammation. Mucosal Immunology, 14(1): 113-124. https: //doi.org/10.1038/s41385-020-0296-4

[24] Shao, M., & Zhu, Y. (2020) Long-term metal exposure changes gut microbiota of residents surrounding a mining and smelting area. Sci Rep, 10(1): 4453. https: //doi.org/10.1038/s41598-020-61143-7

[25] Liu, W., Feng, H., Zheng, S., Xu, S., Massey, I. Y., Zhang, C., Wang, X., & Yang, F. (2021) Pb Toxicity on Gut Physiology and Microbiota. Front Physiol, 12: 574913. https: //doi.org/10.3389/fphys.2021.574913