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THE EFFECT OF POLYSTYRENE FOAM ON THE WHITE MICE'S INTESTINAL MICROBIOTA

Millions of tons of microplastics get into the environment, being eaten by many species of mammals and humans. One of the main types of plastic, polystyrene, and its monomer, bisphenol, have been fairly well studied in terms of their effects on metabolism, but changes in the intestinal microbiota under the influence of its addition to the diet remain insufficiently studied. ***The aim*** *of this article is to describe the changes in the main components of the mice intestinal microbiota in the conditions of adding different concentrations of crushed polystyrene foam to their diet.* ***Methods****. Four groups of white laboratory mice ate crushed particles of polystyrene foam (10% of the polymer by weight of the feed, 1%, 0.1%, and the control group — without addition of plastic) as part of the compound feed for 42 days. At the end of the experiment, cultures of animal feces samples were analyzed.* ***Results****. Polystyrene foam particles in the main mice diet, especially at a higher concentration (10%), have changed the number of the main representatives of the obligate (Bifidobacterium spp.) and some elements of the facultative microorganisms (Lactobacillus spp. and typical Escherichia coli). In all groups of mice that consumed polystyrene foam, there was observed a change in the quantitative ratio of E. coli with normal and altered enzymatic properties. In laboratory animals, to the diet of which 1% or 10% polystyrene foam was added, a decrease in the number of facultative microorganisms was revealed in representatives of the genera Lactobacillus and Enterococcus along with an increase in the number of Pseudomonas aeruginosa bacteria and fungi of the genus Candida.* ***Conclusions****. Such changes can contribute to the reproduction of facultative opportunistic microorganisms and the development of various diseases.*

Keywords: *polymer, plastics, polystyrene, pollution, gut microbiota, dysbiosis mice.*

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In recent years, many researchers have begun to understand the global dependence on polymers used, as they are made from molecular chains that are too elastic to biodegrade in a short period of time [1]. The widespread and universal use of plastic has a negative impact on the environment [2—4]. According to Jambeck et al. [5], 4.8—12.7 million tons of plastic waste have entered the ocean since 2010. Zubris & Richards [6] report unintentional soil contamination with small plastic fragments from sewage sludge, Brinton [7] points out that plastic and glass fragments contaminate compost, while Thompson et al. [8] stress on the plastic that is carried into streams, rivers, and finally into the seas by rainwater and floods. The harm from the plastic can be due to the leaching of monomers, plasticizers, solid additives, and other harmful substances from plastic products (a chemical method) and the breakdown of large particles into micro- or nanoplastics in the environment (a physical method) [9].

Polystyrene foam is a petroleum-based plastic made from styrene monomer, which is used for a variety of packaging, construction, and household purposes. Despite the benefits, since its first commercial production in 1930, the harmful effects of its use have outweighed the benefits of the cheap and convenient use of this plastic type [10, 11].

The production of polystyrene foam pollutes the air and generates large amounts of liquid and solid waste [10, 12]. Thompson et al. [13], Farrelly & Shaw [14], and Turner [15] state that a significant amount of marine litter is polystyrene foam which occurs in air and water, especially along the banks of water bodies. This affects animals: broken pieces of polystyrene block their airways and cause the development of cancer and digestive problems [16].

Lambert & Wagner [17] determined an increase in the formation of nanoplastics during the degradation of polystyrene disposable coffee cup lids. They state that after 56 days of exposure, the concentration of nanoplastics in the polystyrene sample was $1.26 \cdot 10^8$ particles/mL (average particle size 224 nm) compared to $0.41 \cdot 10^8$ particles/mL in the control.

The macroorganism gut microbiome is a complex community of microorganisms that have co-evolved with their host and play a fundamental role in many aspects of its physiology and health. The microbiome composition is individual, but it can vary and depends on various factors (age, gender, diet, as well as the influence of numerous xenobiotics such as pesticides, drugs, amines, salts of heavy metals, etc.). Exposure to xenobiotics can change the intestinal microbiota and its mucosal layer and lead to changes in the metabolic activity. All this can increase the predisposition to various diseases [18—23].

In experiments on mice, Lu et al. [22] have established the effect of polystyrene particles on the microbiota of the caecum contents: there was a significant change in the number of 12 bacterial genera. After 5 weeks of exposure to 0.5 and 50 μM polystyrene, the abundance of *Oscillospira* and *Anaerostipes* decreased, while the abundance of *Parabacteroides, Prevotella, Dehalobacterium, Ruminococcus, Bilophila, Bifidobacterium, Adlercreutzia, Plesiomonas, Halomonas* and *Acinetobacter* increased.

Jin et al. [23] reported a significant change in the abundance of 15 bacterial genera in the caecum contents of mice. It was found that 6 weeks of consuming polystyrene particles (concentrations of 100 and 1000 μg/L) with water led to a significant decrease in *Parabacteroides*, *Prevotella, Dehalobacterium, Turicibacter, Bifidobacterium, Phascolarctobacterium, Lachnospira, Haemophilus, Adlercreutzia, Megamonas, Blautia, Dialister* and *Veillonella*, while the amount of *Coprococcus* and *Anaeroplasma* increased.

Tamargo et al. (2022) [24] have shown that the increase in microplastics in food and beverages alters the composition of the gut microbiome, promoting the biodegradation of plastic particles through digestion and gut bacteria. Polyethylene terephthalate (PET) microparticles in food can alter the composition of the human colon micro-

bial community, so Tamargo et al. [24] suggested that some members of the colonic microbiota can be attached to the surface of microparticles and contribute to the formation of biofilms on them.

Galloway [25] suggests that by 2050 an additional 33 billion tons of plastic will have been added to the planet, which is highly resistant to degradation and is a risk factor for human health and the environment.

However, issues related to the harmful effects of micro- and nanoplastics of various origins on the health of humans and vertebrates remain insufficiently studied. Hence, further work is required to establish the possible consequences for their health. Therefore, **the purpose** of this research is to determine the effect of polystyrene foam on the intestinal microbiota of laboratory animals.

Materials and methods. The study protocol was approved by the local ethics committee of the Dnipro State Agrarian and Economic University. The studies were carried out on the basis of the clinic and laboratories of this university. As experimental animals, white mice were selected and fed for 42 days with crushed particles of expanded polystyrene added to the main diet (Table 1).

The control (first) group of animals was fed only with the basic diet and was given clean water without restriction. To the main diet of the second group animals, 0.1% crushed polystyrene foam was added; for the third group — 1% crushed polystyrene foam; for the fourth group — 10% crushed polystyrene foam (Fig. 1). The grain mixture and rusks were crushed in a mill to the state of flour. Then milk, yeast, fish, and bone meal were added, and granules were formed and dried. Greens and carrots were given separately.

Table 1. **The composition of the diet of experimental animals**

Product	Amount, g
Grain mix (wheat:barley:corn 3:1:1)	5.0
Wheat bread (rusks)	1.3
Oat groats	2.0
Dried milk	2.0—4.0
Fish flour	0.2
Feed yeast	0.1
Bone meal	0.2
Greens (grass)	2.0
Succulent feed (carrots)	2.0

Within 10 days before the experiment, white mice were adapted to the place of their detention and diet. During the study, the dryness of the litter was monitored. At the end of the experiment, all animals were euthanized.

Following the rules of asepsis, after cutting the intestines, samples of animal feces were taken into sterile bottles, sterile saline (1:9) was added, and serial dilutions were made up to 10^{-11} [26].

After all dilutions were prepared, 0.1 mL of suspension was taken from each test tube with a sterile pipette and added to a Petri dish with the appropriate elective medium (Bifidobacterium Agar (HiMedia, India), lactobac agar, *Enterococcus* agar, Endo's medium, bismuth sulfite agar, Wilson & Blair medium, Baird-Parker agar, Sabouraud dextrose agar (OOO Farmaktiv, Ukraine), and 5% blood agar (Biomerieux, France). The suspension was rubbed over the surface with a spatula until it was completely absorbed by the medium and placed for cultivation at 24, 37, and 43 °C for 24—72 hr [27].

Anaerobic conditions for bifidobacteria, lactobacilli, and clostridia were achieved in anaerostats (7 L) using GENbox anaer anaeropackets (Biomerieux, France). Control of anaerobiosis was performed using an Anaer Indicator (Biomerieux, France). CFU/g (colony forming units per 1 gram of intestinal contents) were counted for all Petri dishes [26]. Identification and differentiation of the isolated microorganisms were carried out by studying their enzymatic properties on the Hiss's media with various sugars, Olkenitskyi, Christensen, Simmons citrate agar, malonat agar, etc. (OOO Farmaktiv, Ukraine), as well as using tests API 20 REF 20 600, API Staph REF 10 20 500, API 20 E REF 20 100 / 20 160, API 20 NE REF 20 050, API Candida REF

10 500 (Biomerieux, France) taking into account their biological properties according to the Bergey's Manual of Systematic Bacteriology (1986). Morphology and tinctorial properties were studied under the immersion system of a MICROmedXS-3330 microscope.

Samples were compared using ANOVA with the Bonferroni correction. Data are presented as mean ± standard error (x ± SE).

Results. In 100% of white mice of the control and experimental groups, the base of the intestinal microbiome was anaerobic saccharolytic bacteria of the genera *Bifidobacterium* and *Lactobacillus*. In animals of the control group, the number of probiotic strains of bifidobacteria (mainly 10^{10}) and lactobacilli (10^{10}—10^{11}) corresponded to the reference values of the fecal biopsy of white mice.

In the control group of mice, strains of typical *Escherichia coli* (10^7—10^8 CFU/g) and *E. coli* with altered enzymatic properties (up to 10% within the acceptable range) were isolated; lactose-negative strains formed single colonies. There were also identified other representatives of conditionally pathogenic microorganisms that take part in the formation of intestinal microbiocenosis, namely *Enterobacter* spp., *Citrobacter* spp. (10^2—10^4 CFU/g), *Klebsiella* spp. (10^2 CFU/g), *Proteus* spp. (10^2—10^3 CFU/g), *Enterococcus* spp. (10^7—10^8 CFU/g), *Clostridium* spp. (10^4 CFU/g), *Pseudomonas aeruginosa* (10^2 CFU/g), *Staphylococcus* spp. (10^2—10^3 CFU/g), and *Candida* spp. (10^2 CFU/g). The quantity of these microorganisms corresponds to the reference levels. We did not find representatives of pathogenic microbiota (*Shigella* and *Salmonella*) and hemolytic strains of bacteria (Table 2).

The qualitative composition of the intestinal microbiome of white mice, to which diet a 0.1%

Table 2. **The number of microorganisms (Lg CFU/g of feces) in four groups of mice fed with polystyrene foam particles (x ± SE, n=6, t = 42 days; BD — basic diet))**

Intestinal microbiota	Norm	BD without polystyrene foam	BD + 0.1% of shredded polystyrene foam	BD + 1% of shredded polystyrene foam	BD + 10% of shredded polystyrene foam
Bifidobacterium spp.	8—10	10.60 ± 0.20	10.40 ± 0.20	10.20 ± 0.16	9.80 ± 0.16
Lactobacillus spp.	5—11	10.72 ± 0.16	10.52 ± 0.43	9.44 ± 0.46*	8.68 ± 0.84*
Escherichia coli (normal enzymatic properties strains)	7—8	7.80 ± 0.16	6.98 ± 1.45	4.92 ± 1.64	3.62 ± 1.81
E. coli (weakly fermenting strains)	<7	0.94 ± 0.47	3.46 ± 0.89	3.92 ± 1.42*	4.91 ± 1.67*
E. coli (lactose-negative strains)	2	0.66 ± 0.22	0.00 ± 0.00	1.30 ± 0.66	0.40 ± 0.33
Clostridium spp.	4	2.66 ± 0.58	2.48 ± 0.53	2.66 ± 0.67	3.36 ± 0.86
Enterococcus spp.	7—8	7.32 ± 0.26	6.85 ± 0.74	3.40 ± 0.25***	3.87 ± 0.23***
Proteus spp.	2—3	2.92 ± 0.13	2.84 ± 1.01	4.04 ± 0.89	4.72 ± 1.06*
Staphylococcus aureus	2	1.32 ± 0.13	0.93 ± 0.31	0.92 ± 0.31	1.52 ± 0.32
Enterobacter spp.	2—4	1.96 ± 0.43	3.59 ± 1.24	4.03 ± 1.34	4.58 ± 1.22*
Citrobacter spp.	2—4	1.06 ± 0.40	1.72 ± 0.90	1.24 ± 1.01	2.26 ± 0.93
Klebsiella spp.	2	1.02 ± 0.53	1.44 ± 0.82	0.51 ± 0.42	2.88 ± 1.44
Staphylococcus epidermidis	4	4.05 ± 0.45	3.42 ± 0.11	3.50 ± 0.13	3.87 ± 0.06
Pseudomonas aeruginosa	2	0.92 ± 0.33	1.40 ± 0.49	3.46 ± 0.23***	2.32 ± 0.80*
Candida albicans	2	1.36 ± 0.30	1.91 ± 0.04	0.86 ± 0.29	0.68 ± 0.34
Candida spp.	4	1.71 ± 0.10	2.47± 0.92	3.93 ± 0.18***	4.21 ± 0.10***

Note: * — $P < 0.05$, ** — $P < 0.01$, *** — $P < 0.001$ compared with BD without polystyrene foam using ANOVA with the Bonferroni correction.

polystyrene foam was added (the second experimental group), was similar to the control group: *Bifidobacterium* spp., *Lactobacillus* spp., *Proteus* spp., *Enterococcus* spp., *Clostridium* spp., *Staphylococcus* spp., including *Staphylococcus aureus*, in quantities that correspond to the norm. In animals of this group, lactose-negative strains of *E. coli* were not isolated at all, and, although not reliably, a trend toward a decrease in the number of typical *E. coli* and *Enterococcus* spp. was observed. At the same time, there was a tendency to increase the number of weakly fermenting strains of *E. coli*, *Enterobacter* spp., *P. aeruginosa*, *Candida* spp.

In laboratory animals of the third and fourth experimental groups, to whose diet 1% and 10% of expanded polystyrene was added, a decrease in the number of facultative microorganisms was revealed, namely representatives of the genera *Lactobacillus* ($P < 0.05$) and *Enterococcus* ($P < 0.001$), as well as an unreliable decrease in the number of obligate *Bifidobacterium* spp. and facultative microorganisms — typical *E. coli*.

In animals of all experimental groups, a change in the quantitative ratio of *E. coli* with normal and altered enzymatic properties was noted. The number of *E. coli* strains with a reduced ability to ferment lactose (weakly fermenting) in the experimental groups of animals was 33%, 38%, and 55%, which exceeded the permissible norm (25%), in contrast to the control group (10%). Lactose-negative strains of *E. coli* were found in the form of single colonies or in an acceptable amount.

There was an increase in the number of opportunistic enterobacteria of the genera *Enterobacter* (4.58 ± 1.22 CFU/g compared with 1.96 ± 0.43 CFU/g in animals of the control group, $P < 0.05$) and *Proteus* (4.72 ± 1.06 CFU/g compared with 2.92 ± 0.13 CFU/g in animals of the control group, $P < 0.05$) with the addition of 10% polystyrene foam to the main diet.

Polystyrene foam particles in the amount of 1% and 10% of the daily diet weight contributed to an increase in the abundance of *Pseudomonas aeruginosa* to 3.46 ± 0.23 and 2.32 ± 0.80 CFU/g ($P < 0.05$ and $P < 0.001$, respectively) compared with 0.92 ± 0.33 CFU/g in the control, and yeast-like fungi *Candida* spp. — 3.93 ± 0.18 and 4.21 ± 0.10 CFU/g versus 1.71 ± 0.10 CFU/g in the control group ($P < 0.001$). However, no significant differences were found in the amount of *Candida albicans*, *Staphylococcus epidermidis*, *Staphylococcus aureus,* and *Clostridium* spp. Also, no pathogenic microbiota (*Shigella* and *Salmonella*) and hemolytic strains of bacteria were detected.

Discussion. From 1950 to 2017, a total of 9.2 billion tons of plastic products have been made, which are an essential part of our daily lives, but plastic residues are found both in the environment and in macroorganisms [1, 28—31]. Plastic that was ingested by animals leads to gastrointestinal obstruction [32], intestinal ulceration [33], intestinal perforation, and death [34].

Many authors are concerned about the disposal of plastic waste, which is a potential food chain contaminant [13, 25, 35, 36]. This is especially true for the presence of microscopic plastic debris, or microplastics (debris < 1 mM in size) in aquatic, terrestrial, and marine habitats. Dubaish & Liebezeit [37] and Galloway [25] report the presence of microplastics, and Zubris & Richards [6] — of synthetic polymer fibers, which were found five years after t getting into the soil with settled sewage. According to Cole et al. [38], the main constituent of anthropogenic marine litter is microplastics, consisting of small plastic items such as exfoliators in cosmetics or fragments from larger plastic debris, including polyester fibers from fabrics, plastic bag fragments, and polystyrene particles from buoys and floats.

The results of our previous studies have confirmed the presence of both direct and indirect effects on the gut microbiota by adding plant components to the diet of laboratory animals [39], pesticides and food additives [40, 41], or various types of plastic [42]. Significant changes in the microbiota can occur both directly due

to acting on bacterial cells, and when modeling the immune response of a macro-organism or changes in the activity of the liver, pancreas, and immune cells. The most probable reason is the mechanical impact of plastic particles on the endothelium of the intestinal wall, the violation of the endothelium integrity, the initial stage of the immune response of the macroorganism to the contact with intestinal microorganisms and the transformation of the intestinal microbiota in return to this immune response. The physiological aspects of this interaction between microplastics, immune cells, and hundreds of types of microorganisms have been studied fragmentarily [42].

The polystyrene foam used in our experiment may contain insufficiently polymerized monomers in its composition — bisphenol impurities, obtained in the condensation reaction of two phenol molecules with one acetone molecule. Bisphenol is able to interact with estrogen and other steroid hormone receptors in the body of mammals and humans, which in general causes a weakening of the immune response [43]. Stimulation of immunity by mechanical damage of intestinal endothelial cells by plastic particles, on the one hand, and inhibition of the immune response by the interaction of bisphenol with steroid hormone receptors, on the other hand, is the cause of a complex pattern of metabolic changes [42] and changes in the composition of the microbiota of animals in our experiment.

Jani et al. [44] and Florence & Hussain [45] reported that 50—100 nm polystyrene microspheres are more readily absorbed through Peyer's patches and intestinal villi than larger particles (300—3000 nm). Polymer microparticles are able to retain their chemical composition even when they are mechanically broken into small fragments. Plastic particles are similar to natural substances, they can be ingested by marine and freshwater animals (e.g. turtles, birds, fish, crustaceans). The microplastic then enters the human body through the food chain [13, 38, 46, 47]. Van Cauwenberghe & Janssen [48] found that farmed mussels had higher concentrations of microplastics (178 microfibers) than wild mussels (126 microfibers).

Chen et al. [49] showed that microplastic particles can alter the degradation pathways of decabromodiphenyl ether (BDE-209) and increase its endocrine and thyroid toxicity in aquatic organisms. Endocrine disorders were aggravated by polystyrene foam microparticles covered with biofilms: the content of triiodothyronine increased by 1.7 times, and the expression of thyroid-stimulating hormone beta (TSHB) in zebrafish larvae increased by 5.9 times.

Lusher et al. [50] studied 10 fish species (504 specimens) from the English Channel and found plastic in the gastrointestinal tract in 36.5% of them. Microplastic-contaminated coastlines pose a new threat to the health of Red Sea fish and seafood eaters [51]. In 26 commercial and non-commercial fish species from four different habitats, 26 microplastic fragments were found: 16 films (61.5%) and 10 fishing threads (38.5%). FTIR analysis found that the most common polymers are polypropylene and polyethylene. In the estuary of the Mondego (Portugal), Bessa et al. [52] found 157 microplastics in 38% of the studied fish (fibers in 96% of cases, 1.67 ± 0.27 (SD) microplastics per fish); the predominant polymers identified by μ-FTIR were polyester, polypropylene, and viscose (a semi-synthetic fiber).

Tanaka & Takada [53] examined the digestive tracts for microplastics in 64 species of Japanese anchovies *Engraulis japonicus* collected in Tokyo Bay. Plastic was found in 49 of 64 fish (77%), with an average of 2.3 per fish (52.0% of polyethylene and 43.3% of polypropylene). Most of the plastic was in the form of fragments (86.0%), but 7.3% were balls, some of which were microbeads, similar to those used in cosmetics. In addition, 80% of the released plastic sized between 150 and 1000 μM, which is smaller than the reported size range for floating microplastics on the sea surface.

Polystyrene particles at a dose of 500 μg/mL are not toxic to human cells, and particles with a

diameter of 10—100 μM did not show significant cytotoxicity [54]. However, smaller particles of polystyrene (diameter 460 and 1000 nm) affect erythrocytes.

The toxicity of polystyrene microparticles to terrestrial organisms has been studied less well than to marine organisms. In experiments on male mice, Jin et al. [23] established the effect of polystyrene particles (5 μM in size) on the reduction of intestinal mucus secretion and impaired intestinal barrier function. In addition, due to high-throughput sequencing of the V_3-V_4 region of the ^{16}S rRNA gene, a decrease in Actinobacteria in the caecum content of animals after exposure to polystyrene was revealed: there was a significant change in the number of 15 bacterial genera. Metabolic disorders have been noted in mice as a change in the concentration of amino acids and bile acids in the blood serum [23].

Fackelmann & Sommer [55] suggested that the development of intestinal dysbiosis may be associated with mechanical damage of the gastrointestinal tract due to the ingestion of foreign and potentially pathogenic bacteria, as well as chemicals that are part of microplastics, into the animal's body. In turn, dysbiosis can affect the host's immune system, cause the onset of chronic diseases, contribute to the development of infections caused by pathogenic microorganisms, and alter the expression of the genes of intestinal microorganisms.

Two weeks of exposure to polystyrene particles in adult zebrafish increased their intestinal mucin secretion at a concentration of 1000 μg/L (0.5 μm particles about $1.456 \cdot 10^{10}$ particles/L and 50 μM particles $1.456 \cdot 10^4$ particles/L). Also, it led to dysbiosis of the intestinal microbiota: the number of *Bacteroidetes* and *Proteobacteria* decreased significantly, while the number of *Firmicutes* increased significantly, and intestinal inflammation was observed [56]. These researchers reported changes in the qualitative composition of intestinal microbes and an increase in the level of IL1α, IL1β, and IFN mRNA, as well as their protein concentration in the intestine (after exposure to 0.5 μM polystyrene particles).

Lu et al. [22] exposed male mice to polystyrene (0.5 and 50 μM) for 5 weeks. Oral ingestion of 1000 μg/L of 0.5 and 50 μM polystyrene particles into mice reduced their body weight, liver weight, and blood lipid concentrations. Also, in both experimental groups, there was a decrease in the secretion of mucus in the intestine. Polystyrene exposure reduced the relative abundance of *Firmicutes* and α-*Proteobacteria* in feces. High-throughput sequencing of the V_3-V_4 region of the ^{16}S rRNA gene in the experimental groups, under the influence of 0.5 and 50 μM polystyrene, revealed a change in the microbiome composition : 6 and 8 types of bacteria, respectively, as well as 310 and 160 types of intestinal microorganisms. In addition, animals treated with both 1000 μg/L 0.5 μM and 50 μM polystyrene had reduced levels of hepatic triglycerides (TG) and total cholesterol (TCH). In the liver and epididymal fat, a decrease in the relative level of mRNA of some key genes was noted, associated with lipogenesis and triglyceride synthesis.

Similar results were obtained by Huang et al. [57], who exposed microparticles (diameter 32—40 μM) of polystyrene to juvenile guppies (*Poecilia reticulata*) at 100 and 1000 μg/L for 28 days and found that these particles can exist in the intestines of guppies and cause an increase in goblet cells. While Huang et al. [57] observed a deterioration in digestive function due to the decrease in the activity of digestive enzymes (trypsin, chymotrypsin, amylase, and lipase). Polystyrene microparticles stimulated the expression of immune cytokines (TNF-α, IFN-γ, TLR4, and IL-6) and also caused depletion of the species composition of the guppy gut microbiota.

Xie et al. [58] found that spherical polystyrene microparticles (8 μM) and nanoparticles (80 nm) at a concentration of 1 mg/L for 21 days led to a significant increase in the number of *Proteobac-*

teria and a decrease in Fusobacteria, Firmicutes, and Verrucomicrobiota. The relative number of *Aeromonas* increased significantly in both the microplastic and nanoplastic groups treated. It was also observed that polystyrene nanoparticles are able to induce greater microbiota PAR and cause inflammation in the zebrafish gut.

Esra Tat [1] has reported that tackling plastic requires a holistic approach, as recycling alone cannot solve the plastic crisis. In Europe, a movement called «Zero Waste» has been created, the purpose of which is to stop the waste wave on the planet. An integrated approach is to maximize the use of materials, sort different types of waste, encourage the reduction of polymer production, etc. (to recycle, replace the sale of water in plastic bottles, install public drinking fountains, reuse recycled shoes, clothes, and toys, ban plastic shopping bags, subsidize washable diapers, etc.). Such measures will reduce environmental pollution and the intake of polymers and polystyrene by mammals and will not change the composition of the intestinal microbiota, intestinal barrier, and metabolism.

Conclusions. Polysterene foam particles in the main diet of laboratory mice, especially at a high (10%) concentration, change the number of the main representatives of the obligate (*Bifidobacterium* spp.) and some elements of the facultative (*Lactobacillus* spp. and typical *Escherichia coli*) microorganisms. Such changes can contribute to the reproduction of facultative opportunistic microorganisms and the development of various diseases.

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ВПЛИВ ПІНОПОЛІСТИРОЛУ НА МІКРОБІОТУ КИШЕЧНИКА БІЛИХ МИШЕЙ

Мікропластик мільйонами тон надходить у навколишнє середовище, потрапляючи в їжу багатьох видів ссавців та людини. Один з основних видів пластику — полістирол та його мономер бісфенол достатньо добре досліджені в плані впливу на обмін речовин, однак зміни мікрофлори кишечника за додавання їх в раціон залишаються недостатньо вивченими. **Мета** статті — описати зміни основних компонентів кишкової мікрофлори мишей в умовах додавання до їх раціону різних концентрацій подрібненого пінополістеролу. **Методи.** Чотири групи білих лабораторних мишей упродовж 42 діб поїдали у складі комбікорму подрібнені частинки пінополістиролу (10%, 1%, 0,1% полімеру від маси корму та контрольна група — без додавання пластику). Наприкінці експерименту аналізували посіви проб фекалій тварин. **Результати.** Частинки пінополістиролу в основному раціоні мишей, особливо у найвищій (10%) концентрації, змінили кількість основних представників облігатної (*Bifidobacterium* spp.) та деяких елементів факультативної мікрофлори (*Lactobacillus* spp. та типової *Escherichia coli*). У всіх груп мишей, які споживали пінополістерол, відзначено зміну кількісного співвідношення *Escherichia coli* з нормальними та зміненими ферментативними властивостями. У лабораторних тварин, яким до раціону додавали 1% та 10% пінополістеролу, виявлено зниження кількості факультативної мікрофлори: представників роду *Lactobacillus* та *Enterococcus*, а також збільшення кількості бактерій *Pseudomonas aeruginosa* та грибів роду *Candida*. **Висновки.** Такі зміни можуть сприяти розмноженню факультативних умовно-патогенних мікроорганізмів та розвитку різних захворювань.

Ключові слова: *полімер, пластмаса, полістирол, забруднення, мікробіота кишечника, дисбактеріоз мишей.*