Transactions on Materials, Biotechnology and Life Sciences
ISSN: 2960-1932, eISSN: 2960-2319 | Volume 7
MISBP 2024

Toxicity and mechanism analysis of microplastics

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Abstract. Plastic product the use of plastic product in our daily life led to the potential risk of microplastic exposure and its health outcome. Recently, much research about the toxicity of microplastic in different animals. The result of the toxicity of microplastic in animals may help people find out treatments and assets the environmental effect of the microplastic. This research concludes the experiment of microplastic exposure in different animals and from different pathways, where the animals include the Litopenaeus vannamei, Daphnia manga, Eisenia fetida, zebrafish, human trophoblasts and rats and pathways include cells's oral exposure to the microplastic. The study of microplastic in Litopenaeus vannamei shows the microplastic may inhibit shrimp's immunity in recognizing disease-causing molecules. In addition, the mechanism of toxicity of microplastics will also be analyzed. The research of the recent study of microplastic toxicity to human and animals can help researchers to evaluate the further impact of microplastic pollution to ecosystem, and treatment to microplastic exposure.

Keywords: microplastic; reproductive toxicity; trophoblasts; mechanisms.

1. Introduction

The plastic has played a significant role in our life, from plastic bottles to the chemical fibers that in clothes. Modern organic polymers replaced the traditional materials in many fields thanks to its irreplaceable properties: stable chemical properties, cheap price and abundant usefulness. The plastic production globally has raised rapidly since the 1950 [1] and expected to reach 33 billion tones by 2050. The increase in the plastic production and use lead to the increase in plastic waste [2]. These plastics wastes that remain in environment, will not breakdown as the traditional materials like plant materials. Due to the stable chemical property, plastics wastes may decompose physically instead, become microplastics, which stands for "microscopic plastic fragments and fibers" in 2004 [3]. The presence of microplastics in biosphere has long been scientifically recognized as threat [4-6]. Microplastics in biosphere may expose to human through ingestion, inhalation, and dermal contact, and among these exposure pathways ingestion and inhalation are two major pathways [7]. The microplastics may deposit in organs and cause toxicity [8].

There are many researches uncover the threat that microplastic may bring to human, and the threats mainly come from the damage cause by the accumulation in organs. One example for this is the goldfish research, where this research has explored fish's cell from different organ to microplastic [9]. The results show that microplastic exposure cause the necrosis in gold fish's liver and intestinal liver. Cheng et. al. done research about the toxicity of microplastic and dissociated human embryonic stem cell line H1 (H1 ES) that differentiated toward hepatic lineage expose to microplastic and measure the impact on the cell's viability. The research turns out that the microplastic will be disrupt by certain type of micro plastic, which means the microplastic may exhibit hepatotoxicity [10]. In addition, there is a potential threat that microplastic may pose to cardiovascular system. The research was carried out by Veneman et al., where they inject polystyrene, a part of the microplastics, to Zebrafish embryos, and the research shows that the polystyrene may cause cardiovascular dysfunction at 0.1 ppm [11]. The research from Zhou et al., give polystyrene eye drops to mice, and the outcome shows that the micro plastics may cause inflammation of the conjunctiva and lacrimal gland and cause

damage to the mice's eyes [12]. To this end, this research will discuss the toxicity and mechanism of action of microplastics here, hoping to provide a new strategy for the study of microplastic toxicity.

2. Toxicity of microplastics

2.1. Toxic effect of microplastic in Litopenaeus vannamei

Duan et al. have detailed research on the toxicity of microplastic in Litopenaeus vannamei [13]. The samples for Litopenaeus vannamei were randomly collected from the shrimp farming pond in South China Sea Fisheries Research Institute's Shenzhen Base. The sample size of Litopenaeus vannamei was 900 and exposed to the main raw materials of plastic products, including polyethylene (PE) polystyrene (PS), polyvinyl chloride (PVC), polypropylene (PP) and polytetrafluoroethylene (PTFE). After 14 days of exposure, samples of the entire intestine and hemolymph were analyzed. The result of the research shows that microplastic exposure may have an effect on gut microbiota, and Haemolymph proteomics. According to the research, the stability of the intestinal microbiota decreases and intestinal microbial diversity are not affected. The result of the research also shows the impacts microplastic cause on Haemolymph proteomics and metabolic functions of the intestinal, the microplastic upregulation of PDCD6IP and FKBP1 in the PTFE groups, which inhibit the activation of T cells. The inhibit shrimp's immunity in recognizing disease-causing molecules and might affect cell proliferation supply of energy substances to the organism by effect the homeostasis of nucleotide metabolism since level of L-malic acid decreased. Another important outcome from this experiment is that vWF, RhoA and Arf1 proteins may help reduce the impact of micro plastic, since the level of vWF, RhoA and Arf1 increased when the cell was exposed to microplastic [13].

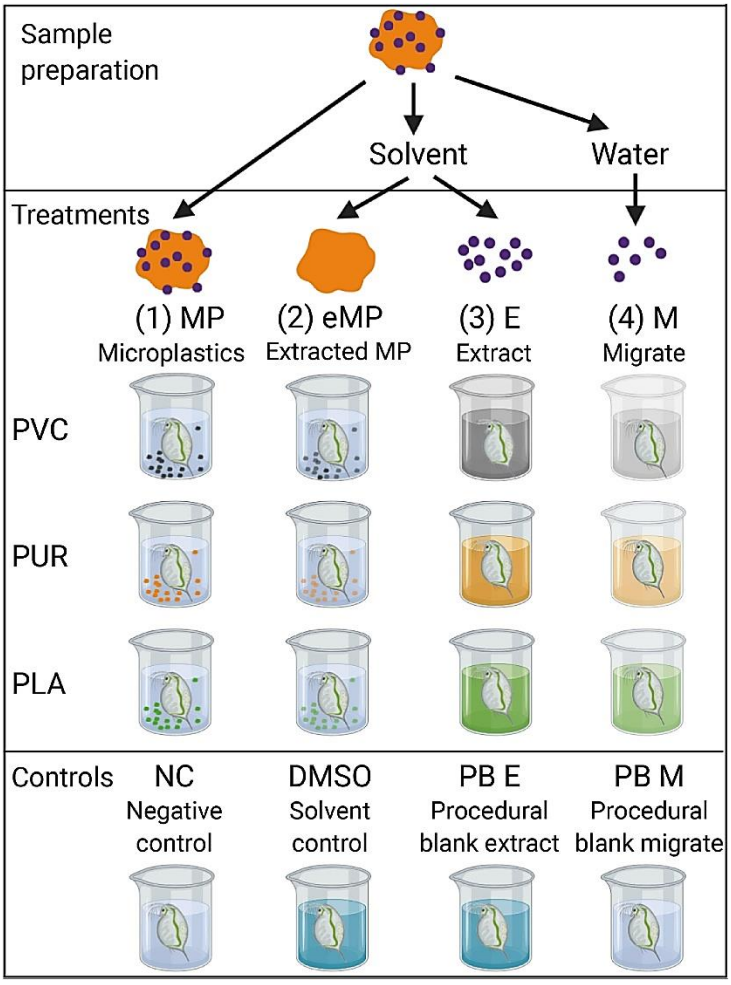

Figure 1. The setting of the experiment for microplastic toxicity [14].

2.2. Toxic effect of microplastic to Daphnia magna

Zimmermann et al. done a study about the Daphnia magna's exposure to petroleum-based microplastic PVC and polyurethane (PUR) and bio-based and biodegradable microplastic polylactic acid (PLA), as shown in Figure 1. The sample size of the Daphnia magna is 10. The sample Daphnia magna then was cultured in 1 L of Elendt M4 medium and feed by live green algae suspension. And the sample microplastics were produced from floor covering, a scouring pad and a shampoo bottle made from different kinds of plastics, rinse frozen and ground in a ball mill until the microplastic particles are in size that can be ingested by Daphnia magna. All kids of microplastic may cause the drop of the number of the offspring of Daphnia magna, PVC drop the offspring number from 101 in the control group to 34 at 100 mg/L, PLA and PUR have similar effect, PLA may drop the number of the offspring to about 9 at 500 mg/L and PUR may decrease the number of the offspring to approximately 40 at 500 mg/L. The exposure of microplastic may also improve the death rate of Daphnia magna and PLA increase the death rate to 60% with concentration-dependent manner, while the control group is 5% although PVC PUR didn't show significant effect on death rate. And the researchers believe that may be the toxicity of microplastic may be driven by plastic chemical [14].

2.3. Toxicity of microplastics on human nasal epithelial cells and rats

Huang et al. done some research about the human nasal epithelial cells and rats' exposure to the microplastic. The experiment used microplastic with 13 different types of functional groups, as shown in Figure 2. According to the outcome of the experiment, the microplastic cell proliferation was inhibited, especially when the concentration of the microplastic higher than 125 μg/mL. For example, at a concentration of 125 μg/mL, the survival rate of HNEpCs exposed to A-PS50 microplastics decreased to about 80% of the control group. Intranasal administration of microplastic of rat may disturb the energy metabolism in body. According to the experiment, the experimental group consumed approximately 55 g and 65 mL of food and water per day after 6 days of administration, while the control group consumed 303.9±4.52 g of food and 402.3±8.20 mL of water [15].

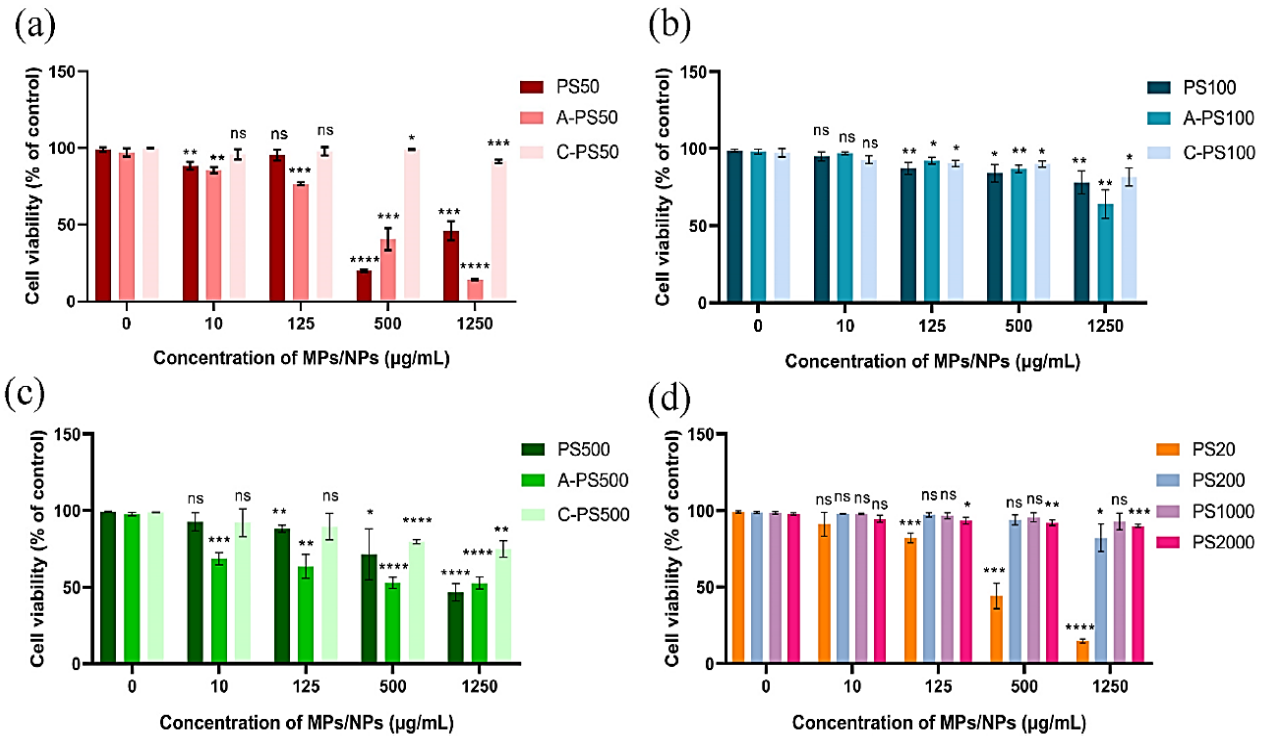

Figure 2. The cell viability of HNEpCs in the presence of different concentrations of microplastics. (a) Different types of PS50. (b) Different types of PS100. (c) Different types of PS500. (d) Different types of PS [15].

2.4. Potential toxicity nanoplastics to human trophoblasts

Hu et al. done researches about the polystyrene nanoplastics's toxicity to human cells *in vitro* [16]. According to the research, the exposure to microplastic lead to inhibition of cell proliferation and probation of apoptosis in HTR-8/SVneo cells. According to the proportion of trophoblasts, the exposure the proportion of cells in S period is 40.18%, which is higher than control group 35.62%. The exposure may cause a cell cycle arrest and delay the transfer of cell from G0/G1 period to S period. The nanoplastics exposure may also decreases trophoblast migration and invasion. According to the Wound-healing assay, the cell invasion of 100 μg/mL group have a cell invasion about 50% of the control group, as the cell migration in 100 μg/mL group was around 83% of the control group. The inflammatory response markers are also observed by ELISA kit, and accumulation of TNF-α from less than 10 pg/mL in control to about 30 pg/ml and IFN-γ from less than 5 pg/mL in control to about 42 pg/mL in cell (pro-inflammatory cytokines) [16].

2.5. Polystyrene nanoplastics toxicity in Eisenia fetida

He et al. done some research about the molecular mechanisms of the toxicity of polystyrene nanoplastics and its mechanisms [17]. In the experiment, the cell viability significantly dropped after contact with polystyrene microplastic. After 24 hours of exposure, the 50 mg/L concentration group's cell viability has dropped to around 75% of the control group, and the cause of this cell viability drop may be the oxidative stress caused by the micro plastic. According to further experiment, the group that pre-treatment with NAC's cell viability (24 hours of exposure the 50 mg/L concentration) rise to around 80% of the control group (Figure 3). Since the oxidative stress is cause by increase in ROS concentration in cells, a failure in immunity system will be caused due to the increased ROS concentration. In 50 mg/L 24 hours group, the ROS level in cell has reached near 190% without NAC, and NAC pre-treatment may decrease the ROS level in cell because the same group dropped to around 180% of control group with NAC pretreatment [17].

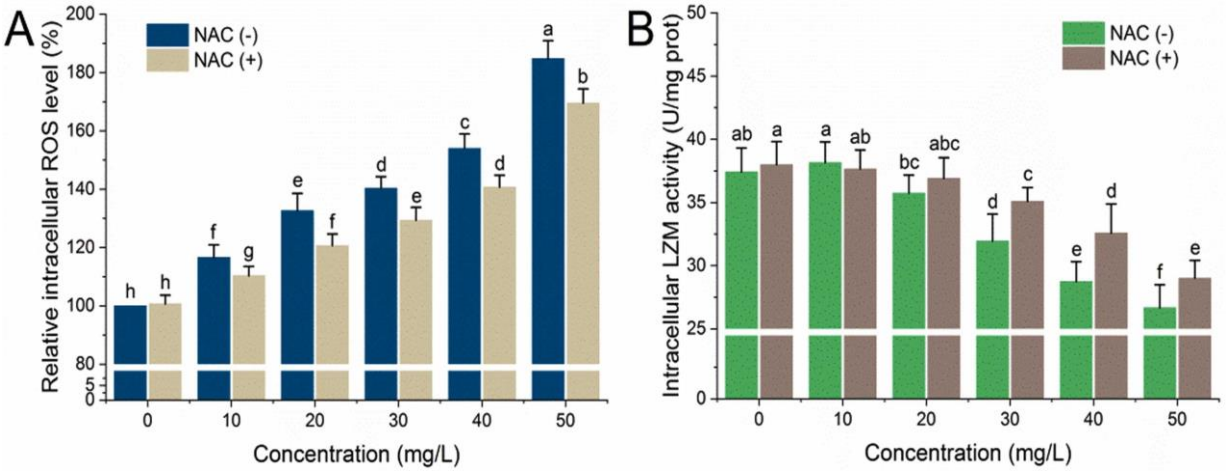

Figure 3. Relative intracellular ROS level (A) and intracellular LZM activity (B) [17].

3. Conclusion

In conclusion, this research did a discussed about the recent study about the toxicity of microplastic and its mechanism. This research also discusses the impact of microplastics on different organisms. The research shows that the microplastic's toxicity to organism is mainly about disturb of the supply of energy substances to the organism, oxidative stress, imbalance in sex steroid hormones and reproductive toxicity. The increase in death rate may cause by the chemical property of the microplastic. The research on microplastic toxicity and mechanisms provides a reference for future in-depth investigations on microplastic.

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