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# The toxicity of polyethylene microplastics on molecular and biochemical parameters in albino mice

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## ABSTRACT

Microplastics (MPs) pose a growing environmental and health threat. Polyethylene (PE), a common plastic, is increasingly accumulating in the environment and organisms, raising concerns about its potential impact on human health. This study investigated PE-MPs' impact on biochemical and molecular markers in adult male albino mice. Three groups of mice were utilized: one group received distilled water as a control. In contrast, the other groups were administered oral gavage treatments of PE-MPs at dosages of 1.3 mg/kg or 0.6 mg/kg every other day for 45 days. PE-MPs significantly increased malondialdehyde (MDA) levels with exposure time, especially at higher doses, inducing significant oxidative stress ($P < 0.021$) compared to the control group. Glutathione peroxidase (GPx) levels increased with exposure time ($P < 0.0393$), especially at lower doses. thus adversely affecting oxidants/antioxidants balance. Moreover, PE-MPs increased T3 and TSH levels significantly elevated in the high-dose group (G1, 1.3 mg/kg) compared to both the low-dose group (G2, 0.6 mg/kg) and the control group. The comet assay confirmed the genotoxic effects on DNA, which were evident through increased DNA damage. This highlights the necessity for more research to examine the health risks associated with environmental microplastics. We can have concluded that**,** the toxicity of microplastics is dose-dependent, indicating that increased exposure leads to heightened harm.

**Keywords:** microplastics, environmental factors, pollutants, oxidative stress, DNA damage, Thyroid hormones, human health risk.

## INTRODUCTION

Plastics have permeated many aspects of our lives, with production increasing substantially in recent decades. Plastics are substitutes for glass, metals, paper, and wood (Yao et al., 2022). By 2025, roughly 100 to 250 million tons are projected to enter surface waters (Ali et al., 2021) nevertheless, increased output results in heightened pollution and, hence, more potential health hazards.(Abdel-Zaher et al., 2023) MPs are tiny pieces of plastic that range in size from 0.1 mm to 5 mm. Polyethene (PE) is one of the most common elements of MPs, along with polypropylene (PP), polystyrene (PS), and polyvinyl chloride (PVC) (Santacruz-Juárez et al., 2021). Although polyethene, polypropylene, and polystyrene microplastics were believed to be primarily located in the oceans (Wei et al., 2021). The two primary sources of microplastics (MPs) are the degradation of bigger plastic particles through UV light, mechanical items include sunscreens, cosmetics, detergents, and medication delivery systems that contain plastic powders, as well as abrasion, biological deterioration, and different environmental conditions (Abdel-Zaher et al., 2023). MPs can accumulate in diverse creatures, including humans. Several studies have shown that MPs can penetrate biological tissues because of their small size and non-biodegradable nature (Jin et al., 2021). It has been documented that MP toxicity affects many organs, including the brain, kidneys, liver, and reproductive system (Kim et al., 2021). Further research is necessary due to the complex pathophysiology of MP toxicity in mammals. The beginning of oxidative stress, cytotoxicity, and inflammation

is associated with MP exposure. They create malfunctions in subcellular organelles and interfere with the metabolism of fats and energy (Llorca and Farré, 2021). An imbalance in both antioxidants and reactive oxygen species (ROS) is known as oxidative stress. Excess ROS could result from a rise in ROS production, a decrease in antioxidants that decreases reactive oxygen species, or combination of the two (Salman and AL-Jumaily, 2024). Hematological parameters and blood analysis serve as effective indicators for assessing harmful substance exposure and an individual's overall health (Falah and Rabee, 2022). One primary concern regarding MPs is their potential impact on DNA and their function as mutagenic and epigenetic contaminants. Increasing apprehension exists regarding the potential genotoxicity to humans caused by microplastics (Çobanoğlu et al., 2021). This study examined the potential molecular consequences of polyethene microplastics (PE-MPs) in mammals. This study seeks to ascertain if exposure to microplastics induces molecular-level alterations and, if so, if these alterations correlate with the quantity of microplastics an organism encounters. We anticipated that exposure to these particles could result in DNA damage, thyroid dysfunction, and oxidative stress abnormalities.

## MATERIALS AND METHODS

### Microplastics

Microplastics made of polyethylene (PE-MPs) were acquired from SABIC Saudi Arabia in powdered was purchased from Areej Al-furat Company (a chemicals company in Baghdad, Iraq), characterized by asymmetrical particles. The diameters vary from (14 nm to 184 nm), with a mean diameter of (135 nm) and a density of (5,954.297) particles per nm$^2$.PE-MP dry powder was suspended in maize oil and thoroughly vortexed before oral gavage in mice (Sun et al., 2021).

### Animal treatment and experimental conditions

We purchased male mice (Mus musculus, ICR) from the National Centre for Drug Control and Research that were five weeks old and weighed 25±27 g. A 12-hour light/dark cycle, consistent humidity and temperature, and unlimited access to food and water were all features of their controlled environment. Every experimental method was carried out in compliance with the ethical standards for research using animals.

The ethical approval of the study was obtained from the Ethics Committee (University of Baghdad, College of Science, Department of Biology, Ref. no. CSEC/1023/0087 on October 13, 2023).

### Experimental scheme

Following a one-week acclimatization period, the mice were categorized into three groups based on the dosage of microplastic polyethene administered. A lethal dose test was used to determine the final experimental dosages, which were chosen based on previous studies (Sun and Wang, 2023). The calculations were based on the body weight of the mice (25 ± 27 g) and the volume of drinking water (4 mL/day). Each group contained ten mice administered oral gavage every two days. This study involved three groups: Group 1, the control group, received distilled water; Group 2 received 1.3 mg/kg of MPs-PE; and Group 3 received 0.6 mg/kg of MPs-PE, all for 45 days, given that the MPs attained a steady concentration in the intestinal tracts of mice following a two-week gavage (Deng et al., 2017).

### Collection of blood samples

Twenty-four hours following the last injection, blood samples were taken from post-mortem hearts. The remainder was centrifuged to extract serum for biochemical investigation, and a part was utilized for the alkaline comet assay. The serum was kept at -20 °C.

### Oxidative stress parameters

The serum concentration of MDA was assessed using the Buege and Aust technique (Alam et al., 2013). MDA, resulting from the degradation of polyunsaturated fatty acids, is a reliable indicator of peroxidation reactions. The thiobarbituric acid method was employed to quantify MDA, which interacts with thiobarbituric acid (TBA) to produce a pink hue measured at λ max 535 nm (Buege and SD). Serum glutathione

peroxidase (GPX) levels were quantified using the Mice GPX Elisa Kit (BTLAB, China, Catalog number: E3922Hu), per the manufacturer's guidelines (Ahmed and Yenzeel, 2017).

### Biochemical parameters

ELISA technique was also used to determine serum levels of T3 and TSH were quantified with commercial rat T3 Elisa Kit (KHBIO, China, Catalog number: ER1720) and rat TSH Elisa Kit (KHBIO, China, Catalog number: ER1411) Through the manufacturer's instructions (Gharb et al., 2024).

### DNA damage detected by the alkaline comet assay

The Comet Assay was employed to identify DNA breaks and alkali-labile locations. Blood samples were diluted and combined with agarose on slides to create layers. The slides were subsequently lysed, rinsed, and analyzed by electrophoresis. Following staining with Ethidium Bromide, DNA damage was observed and quantified utilizing a fluorescence microscope and image analysis software (Jaffer and Rabee, 2024).

### Statistical analysis

To assess significant differences among study groups regarding T3, TSH, GPX, MDA, two-way ANOVA followed by Tukey's test were done. The differences were considered significant when P value $\leq 0.05$. The statistical analysis was performed using GraphPad Prism 9.2 (Pagano et al., 2022).

## RESULTS AND DISCUSSION

### Characterization of polyethylene micrplastics

There are numerous polyethylene particles of various sizes and shapes that have significantly polluted the sample surface. There are more small to medium-sized particles than larger ones. The rough and uneven surface suggests that the particles' size and shape were changed by exposure to outside influences. With an unequal distribution and a broad range of point heights, statistical analysis validates the surface's roughness. The roughness observation is further supported by the 3D model, which depicts a rocky, uneven ground (Fig. 1).

### Effect of PE-MPs exposure on oxidative stress and related parameters GPX and MDA

Figure 2 shows the effects of polyethylene microplastic exposure on malondialdehyde (MDA) levels. After six weeks, higher doses (1.3 mg/kg) induced significant oxidative stress ($P < 0.021$) compared to the control group. Glutathione peroxidase (GPx) levels increased with exposure time ($P < 0.0393$), especially at lower doses. The interaction between duration and concentration ($P < 0.0497$, $P < 0.0361$) suggests that the impact of exposure time varies with the dose.

### Effect of PE-MPs exposure on thyroid hormones T3 and TSH

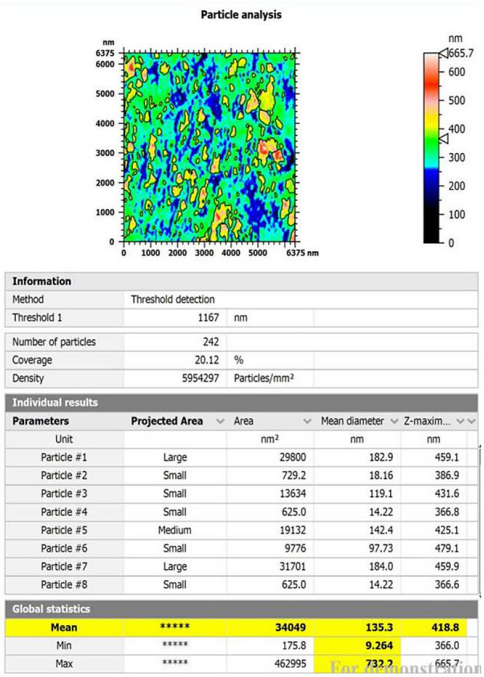
After 3 weeks, T3 levels were significantly elevated in the high-dose group (G1, 1.3 mg/kg) compared to both the low-dose group (G2, 0.6 mg/kg) and the control group ($P < 0.025$, $P < 0.044$). While T3 levels remained elevated in both treated groups after 6 weeks, the reduction was more significant in the high-dose group ($P < 0.0307$) (Fig. 3). ANOVA analysis revealed that duration significantly influenced T3 levels ($P < 0.0031$), while concentration did not have a significant main effect ($P = 0.0582$). There was no significant interaction effect between duration and concentration ($P > 0.8289$), suggesting that their effects on T3 levels are independent.

Figure 4 shows that TSH levels were significantly elevated in both treatment groups (1.3 and 0.6 mg/kg) compared to the control group ($P < 0.0001$). The higher dose group exhibited a more pronounced increase in TSH levels compared to the lower dose group. ANOVA analysis revealed significant interaction effects between duration and concentration ($P < 0.0001$), indicating that the effects of these factors on TSH levels are interdependent. Both duration and concentration were found to have significant main effects on TSH levels ($P < 0.0001$).

### DNA damage induced by exposed to PE-MPs

Figure 5 shows that PE-MPs significantly increased DNA levels in both treatment groups compared to the control group ($P < 0.0001$). A dose-dependent effect was observed, with the higher dose (1.3 mg/kg) inducing a more significant increase in DNA levels ($P<0.0049$). Additionally, a time-dependent effect was evident,

**A.**

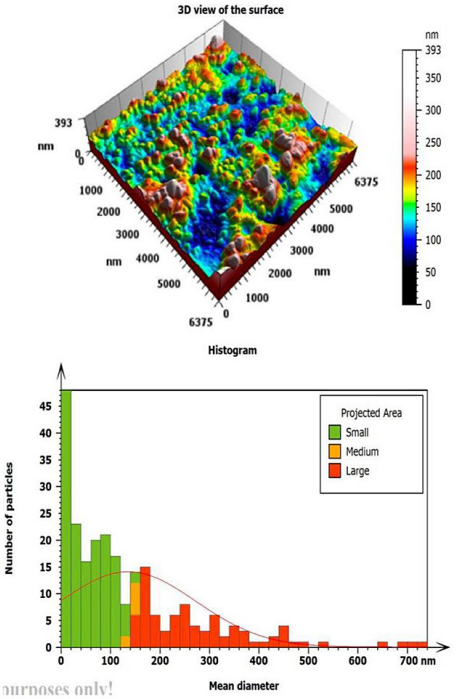



| Information | | |
|---|---|---|
| Method | Threshold detection | |
| Threshold 1 | 1167 | nm |
| Number of particles | 242 | |
| Coverage | 20.12 | % |
| Density | 5954297 | Particles/mm² |

| Individual results | | | | |
|---|---|---|---|---|
| **Parameters** | **Projected Area** | **Area** | **Mean diameter** | **Z-maxim...** |
| Unit | | nm² | nm | nm |
| Particle #1 | Large | 29800 | 182.9 | 459.1 |
| Particle #2 | Small | 729.2 | 18.16 | 386.9 |
| Particle #3 | Small | 13634 | 119.1 | 431.6 |
| Particle #4 | Small | 625.0 | 14.22 | 366.8 |
| Particle #5 | Medium | 19132 | 142.4 | 425.1 |
| Particle #6 | Small | 9776 | 97.73 | 479.1 |
| Particle #7 | Large | 31701 | 184.0 | 459.9 |
| Particle #8 | Small | 625.0 | 14.22 | 366.6 |

| Global statistics | | | | |
|---|---|---|---|---|
| **Mean** | ***** | **34049** | **135.3** | **418.8** |
| Min | ***** | 175.8 | **9.264** | 366.0 |
| Max | ***** | 462995 | **732.2** | 665.7 |

**B.**




**C.**

| ISO 25178 - Primary surface | | | | |
|---|---|---|---|---|
| *S-filter (λs): None* | | | | |
| *F: [Workflow] Surface, form removed (LSP 3)* | | | | |
| **Height parameters** | | | | |
| **Sq** | **48.18** | **nm** | | ***Root-mean-square height*** |
| Ssk | 0.5249 | | | *Skewness* |
| Sku | 4.191 | | | *Kurtosis* |
| Sp | 220.7 | nm | | *Maximum peak height* |
| Sv | 203.0 | nm | | *Maximum pit depth* |
| **Sz** | **423.7** | **nm** | | ***Maximum height*** |
| **Sa** | **37.11** | **nm** | | ***Arithmetic mean height*** |
| **Functional parameters** | | | | |
| Smr | 100.0 | % | *c = 1000 nm below highest peak* | *Areal material ratio* |
| Smc | 63.89 | nm | *p = 10%* | *Inverse areal material ratio* |
| Sdc | 116.7 | nm | *p = 10%, q = 90%* | *Material ratio height difference* |
| **Spatial parameters** | | | | |
| Sal | 510.8 | nm | *s = 0.2* | *Autocorrelation length* |
| Str | 0.7569 | | *s = 0.2* | *Texture-aspect ratio* |
| Std | 176.3 | ° | *Reference angle = 0°* | *Texture direction* |
| Ssw | 27.07 | nm | | *Dominant spatial wavelength* |
| **Hybrid parameters** | | | | |
| Sdq | 0.4029 | | | *Root-mean-square gradient* |
| Sdr | 7.247 | % | | *Developed interfacial area ratio* |
| **Functional parameters (volume)** | | | | |
| Vm | 3.238 | nm³/nm² | *p = 10%* | *Material volume* |
| Vv | 67.13 | nm³/nm² | *p = 10%* | *Void volume* |
| Vmp | 3.238 | nm³/nm² | *p = 10%* | *Peak material volume* |
| Vmc | 38.45 | nm³/nm² | *p = 10%, q = 80%* | *Core material volume* |
| Vvc | 62.70 | nm³/nm² | *p = 10%, q = 80%* | *Core void volume* |
| Vvv | 4.423 | nm³/nm² | *p = 80%* | *Pit void volume* |
| **Feature parameters** | | | | |
| Spd | 2.706e-06 | 1/nm² | *Pruning = 5%* | *Density of peaks* |
| Spc | 0.006326 | 1/nm | *Pruning = 5%* | *Arithmetic mean peak curvature* |
| S10z | 314.1 | nm | *Pruning = 5%* | *Ten point height* |
| S5p | 179.7 | nm | *Pruning = 5%* | *Five point peak height* |

**D.**

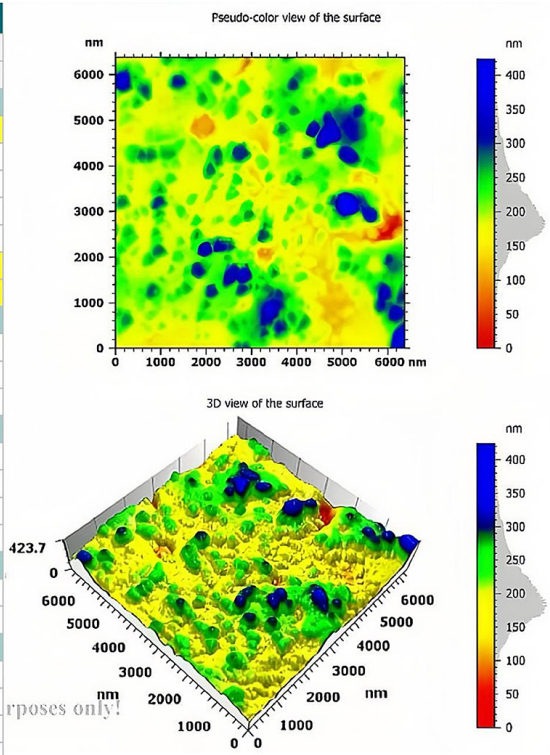



**Figure 1.** (A, B, C, D) presents a detailed table of surface roughness parameters, including maximum height, area, and statistical measures like RMS roughness, skewness, and kurtosis. The 3D image visually illustrates the surface's uneven topography, complementing the numerical data

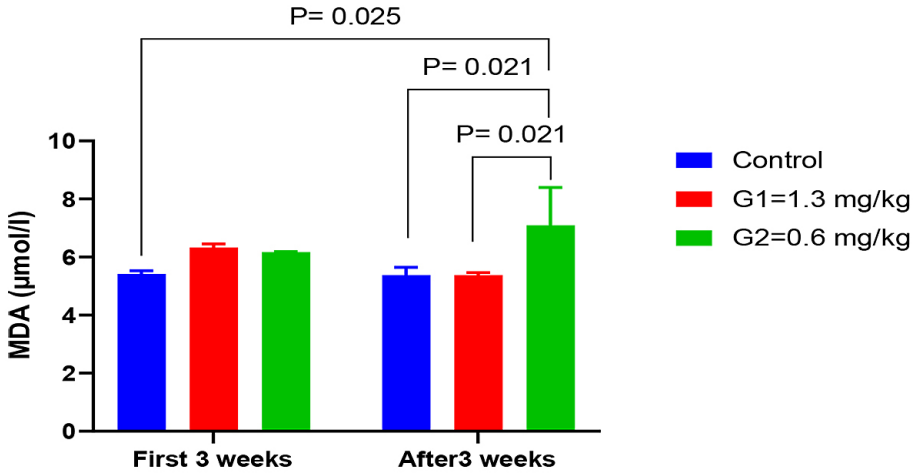



**Figure 2.** PE-MPs (1.3 and 0.6 mg/kg) had no significant effect on GPx or MDA levels after 3 weeks. However, after 6 weeks, both biomarkers were significantly elevated compared to controls ($P < 0.021$; $n = 10$)

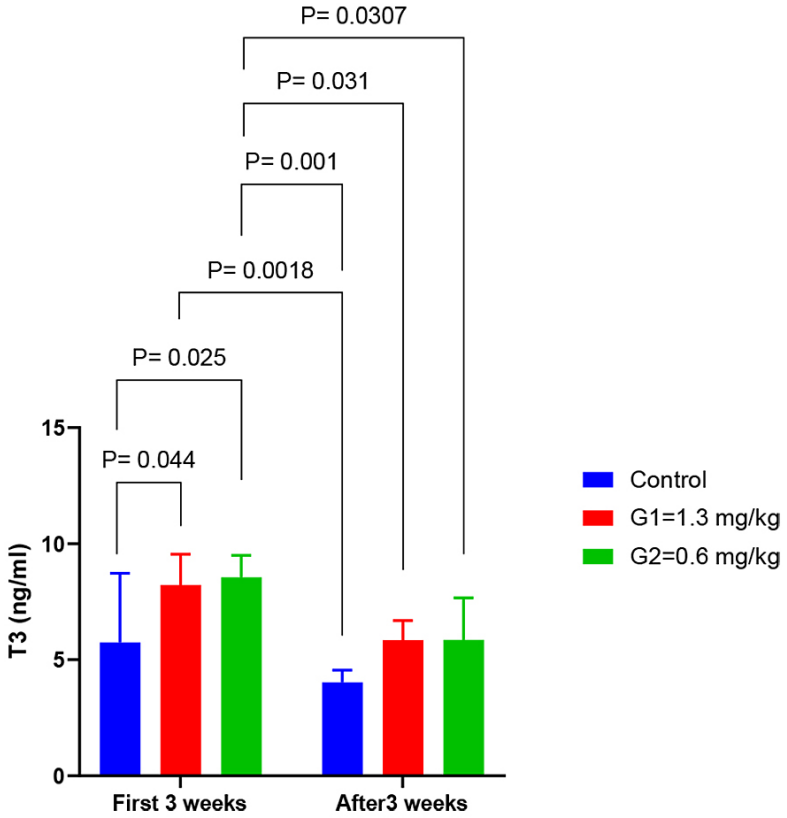



**Figure 3.** The comparison of T3 levels in control and PE-MP-exposed groups (G1: 1.3mg/kg, G2: 0.6 mg/kg) over a certain period of time (after six weeks). n = 10

with DNA levels increasing more significantly after 6 weeks than after 3 weeks ($P < 0.0001$). ANOVA analysis revealed significant effects of both duration and concentration on DNA levels (P value $\leq 0.05$). The interaction between duration and concentration was also significant (P value $\leq 0.05$), indicating that the effect of duration depends on the concentration level.

## Microplastics PE-MPs

Microplastics considered persistent pollutants, are increasingly in our environment, including food, water, and air. Their accumulation in living organisms can lead to various health issues, such as gastrointestinal problems, immune system dysfunction, respiratory diseases, cancer, infertility, and

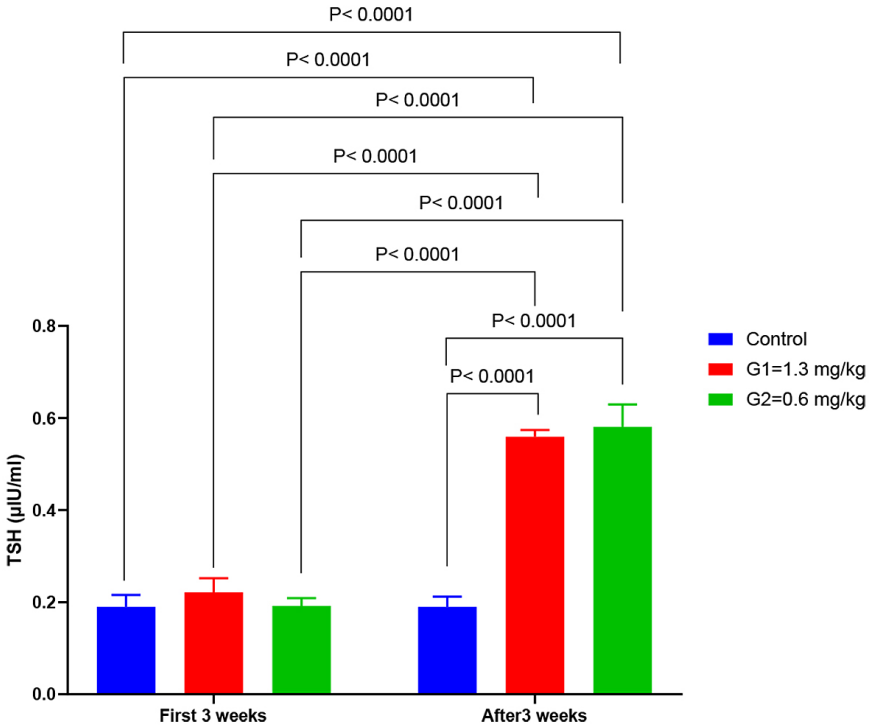



**Figure 4.** Shows that both PE-MP concentrations (1.3 and 0.6 mg/kg) significantly increased TSH levels compared to the control group after 6 weeks (P < 0.0001, n =10)

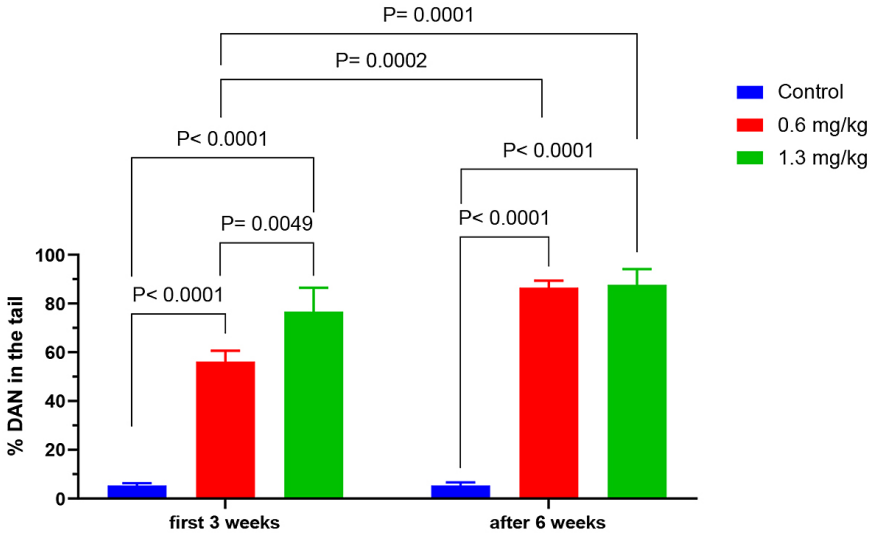



**Figure 5.** PE-MP exposure significantly increased tail DNA levels in both treatment groups compared to controls after 6 weeks (P < 0.0001, n = 10)

genetic abnormalities. Further research is needed to fully understand the health risks associated with microplastic exposure (Haindongo et al., 2023).

MPs function as conduits for various substances and diverse toxicants, leading to several detrimental consequences on the human and other animal physiologies. To yet, the effects of MPs on human health are mostly unknown (Kim et al., 2021). Animal models have been demonstrated to help assess microplastic exposure concerns, which can assist in evaluating their effects on the human body (da Silva Brito et al., 2022). Polyethylene is the predominant type of microplastics in the environment (Sun et al., 2021). Evidence suggests that PE-MPs possess pro-oxidative characteristics (Djouina et al., 2023; Hu and Palić, 2020).

### Effect of PE-MPs on oxidative stress and related parameters GPX and MDA

One way that MPs affect the cell membrane is by increasing the amount of ROS, which can result in direct or indirect harm. MPs physically damage cells when they come into contact with the cell membrane. Many particles can adhere to the membrane adsorptively thanks to hydrophobic interactions and Van der Waals forces (Liu et al., 2021). Higher doses of PE-MPs led to increased oxidative stress, characterized by higher ROS levels and decreased antioxidant defenses, compared to lower doses. These findings align with previous studies (Farag et al., 2023; Li et al., 2022).

### DNA damage

The comet assay, as recommended by ICH S2 (R1), showed that PE-MP exposure caused significant DNA damage, likely due to increased ROS levels, which induce oxidative DNA damage (Roursgaard et al., 2022). Exposure to PET and PP microplastics can cause DNA damage, possibly through direct interaction with DNA rather than oxidative stress. While these findings suggest potential genotoxic effects, they did not reveal direct toxicity. By the present study (Ballesteros et al., 2020), Exposure to PS nanoparticles significantly increased DNA damage in monocytes and PMN cells. Differences in plastic particle type, functionalization, and size may explain discrepancies in study results (Malinowska et al., 2022), can suggest to apply PCR technique to detection the damages in encoding genes that responsible to produced functional proteins, the molecular techniques were used indifferent filed of Biology (Ismael et al., 2023; Ibrahim and Laftaah, 2024; Abdullah and Al-Rubaii, 2024; Sultan et al, 2023).

### Effect of PE-MPs exposure on thyroid hormones T3 and TSH

Our study suggests that microplastics can disrupt thyroid function by affecting the hypothalamic-pituitary axis or directly interfering with thyroid hormone synthesis, secretion, transport, metabolism, or receptor interactions. Significant changes in TSH and T3 levels were observed, particularly at higher microplastic concentrations (1.3 mg/kg). Further research is needed to confirm the statistical significance of the observed T3 reduction (Pathak, 2025). Moreover, research on actual environmental exposures has shown that studies indicating detrimental effects on thyroid hormones often utilize microplastic concentrations that are too elevated (Tang et al., 2024). These findings were congruent with prior studies.

## CONCLUSIONS

This study demonstrates that microplastic exposure in mice can lead to adverse effects, including altered oxidative stress, thyroid dysfunction, and DNA damage. The induction of OS is the basis for the harmful effects that MPs have on the majority of organisms. Increased ROS generation can lead to apoptosis, lipid peroxidation, DNA damage, mitochondrial shape and function loss, and an increase in cellular inflammation. The reduced mitochondrial membrane potential and ROS-induced mitochondrial depolarisation will lead to increased ROS formation, which would exacerbate the previously described damage. MPs therefore affect cells, tissues, organs, and entire organisms. This thorough analysis revealed significant variation in the stated relationship between MPs and OS. These effects were more pronounced at higher doses, highlighting the dose-dependent toxicity of microplastics. This research suggests that microplastics may pose significant risks to human health and the environment. More research is needed, especially on animal models, in order to develop a standardized research technique with a public health focus. Focusing on how detrimental MPs are when mixed with other chemical pollutants and, eventually, on strategies to reduce the harm that MPs and associated OS do to the environment would be advantageous. Due to the growing number of MPs food contamination cases, these themes are especially pertinent.

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## REFERENCES


1. Abdel-Zaher, S., Mohamed, M. S., & Sayed, A. E.-D. H. (2023). Hemotoxic effects of polyethylene microplastics on mice. *Frontiers in Physiology, 14*, 1072797. https://doi.org/10.3389/fphys.2023.1072797
2. Abdullah, M. M., & AL-Rubaii, B. A. L. (2024). Effect of Lactobacillus supernatant on swarming-related gene expression in Proteus mirabilis isolated from urinary tract infections. *Ukrainian Journal of Nephrology and Dialysis*, 4(84), 39-48. https://doi.org/10.31450/ukrjnd.4(84).2024.05
3. Ahmed, A. K., & Yenzeel, J. H. (2017). Determination of some oxidative stress parameters and antioxidants in sample of Iraqi beta thalassemia major patients. *Iraqi Journal of Science*, 1–3.
4. Alam, M. N., Bristi, N. J., & Rafiquzzaman, M. (2013). Review on in vivo and in vitro methods evaluation of antioxidant activity. *Saudi Pharmaceutical Journal, 21*(2), 143–152.
5. Ali, I., Cheng, Q., Ding, T., Yiguang, Q., Yuechao, Z., Sun, H., Peng, C., Naz, I., Li, J., & Liu, J. (2021). Micro-and nanoplastics in the environment: Occurrence, detection, characterization and toxicity–A critical review. *Journal of Cleaner Production, 313*, 127863. https://doi.org/10.1016/j.jclepro.2021.127863
6. Ballesteros, S., Domenech, J., Barguilla, I., Cortés, C., Marcos, R., & Hernández, A. (2020). Genotoxic and immunomodulatory effects in human white blood cells after ex vivo exposure to polystyrene nanoplastics. *Environmental Science: Nano, 7*(11), 3431–3446.
7. Buege, J., & SD, A. (1978). Microsomal lipid peroxidation methods,. *Enzymol., 52*, 302–310. In: ed.
8. Çobanoğlu, H., Belivermiş, M., Sıkdokur, E., Kılıç, Ö., & Çayır, A. (2021). Genotoxic and cytotoxic effects of polyethylene microplastics on human peripheral blood lymphocytes. *Chemosphere, 272*, 129805.
9. da Silva Brito, W. A., Mutter, F., Wende, K., Cecchini, A. L., Schmidt, A., & Bekeschus, S. (2022). Consequences of nano and microplastic exposure in rodent models: the known and unknown. *Particle and Fibre Toxicology, 19*(1), 28. https://doi.org/10.1186/s12989-022-00473-y
10. Deng, Y., Zhang, Y., Lemos, B., & Ren, H. (2017). Tissue accumulation of microplastics in mice and biomarker responses suggest widespread health risks of exposure. *Scientific Reports, 7*(1), 46687. https://doi.org/10.1038/srep46687
11. Djouina, M., Waxin, C., Dubuquoy, L., Launay, D., Vignal, C., & Body-Malapel, M. (2023). Oral exposure to polyethylene microplastics induces inflammatory and metabolic changes and promotes fibrosis in mouse liver. *Ecotoxicology and Environmental Safety, 264*, 115417.
12. Falah, Z., & Rabee, A. M. (2022). The effects of combined toxicity of silver and silicon nanoparticles on hematological and biochemical parameters in male albino mice. *Iraqi Journal of Science*, 4195–4204.
13. Farag, A. A., Youssef, H. S., Sliem, R. E., El Gazzar, W. B., Nabil, N., Mokhtar, M. M., Marei, Y. M., Ismail, N. S., Radwaan, S. E., & Badr, A. M. (2023). Hematological consequences of polyethylene microplastics toxicity in male rats: Oxidative stress, genetic, and epigenetic links. *Toxicology, 492*, 153545. https://doi.org/10.1016/j.tox.2023.153545
14. Gharb, L. A., Ismael, M. K., & Qaddoori, Y. B. (2024). Evaluation of the activity of olibanum oil as an immune booster in rats. *Iraqi Journal of Science.* https://doi.org/
15. Haindongo, N. N., Breen, C. J., & Neretin, L. (2023). Emerging contaminants related to plastic and microplastic pollution. In: *Present Knowledge in Food Safety*, Elsevier, 270–280.
16. Hu, M., & Palić, D. (2020). Micro-and nano-plastics activation of oxidative and inflammatory adverse outcome pathways. *Redox Biology, 37*, 101620.
17. Ibrahim, G. J., & Laftaah, B. A. (2024). The efficiency of certain amino acids in regulating chABCI gene expression in proteus mirabilis. *Iraqi Journal of Science*, 65(9), 4983-4992. https://doi.org/10.24996/ijs.2024.65.9.15
18. Ismael, M. K., Qaddoori, Y. B., Shaban, M. N., & Laftaah AL-Rubaii, B. A. (2023). The immunohistochemical staining of vimentin and E-cadherin in bladder cancer patients infected with hepatitis C virus. *Journal of Pure & Applied Microbiology*, 17(2), 1009–1016. https://doi.org/10.22207/JPAM.17.2.30
19. Jaffer, N. S., & Rabee, A. M. (2024). Combined effects of cypermethrin and lead on biochemical and molecular parameters in albino mice. *Iraqi Journal of Science*, 4912–4920. https://doi.org/10.24996/ijs.2024.65.9.8
20. Jin, H., Ma, T., Sha, X., Liu, Z., Zhou, Y., Meng, X., Chen, Y., Han, X., & Ding, J. (2021). Polystyrene microplastics induced male reproductive toxicity in mice. *Journal of Hazardous Materials, 401*, 123430. https://doi.org/10.1016/j.jhazmat.2020.123430
21. Kim, J.-H., Yu, Y.-B., & Choi, J.-H. (2021). Toxic effects on bioaccumulation, hematological parameters, oxidative stress, immune responses and neurotoxicity in fish exposed to microplastics: A review. *Journal of Hazardous Materials, 413*, 125423. https://doi.org/10.1016/j.jhazmat.2021.125423
22. Li, Z., Chang, X., Hu, M., Fang, J. K.-H., Sokolova, I. M., Huang, W., Xu, E. G., & Wang, Y. (2022). Is microplastic an oxidative stressor? Evidence from a meta-analysis on bivalves. *Journal of Hazardous Materials, 423*, 127211.
23. Liu, L., Xu, K., Zhang, B., Ye, Y., Zhang, Q., & Jiang, W. (2021). Cellular internalization and release

of polystyrene microplastics and nanoplastics. *Science of the Total Environment, 779*, 146523.

24. Llorca, M., & Farré, M. (2021). Current insights into potential effects of micro-nanoplastics on human health by in-vitro tests. *Frontiers in Toxicology, 3*, 752140. https://doi.org/10.3389/ftox.2021.752140
25. Malinowska, K., Bukowska, B., Piwoński, I., Foksiński, M., Kisielewska, A., Zarakowska, E., Gackowski, D., & Sicińska, P. (2022). Polystyrene nanoparticles: the mechanism of their genotoxicity in human peripheral blood mononuclear cells. *Nanotoxicology, 16*(6–8), 791–811. https://doi.org/10.1080/17435390.2022.2149360
26. Pagano, M., Gauvreau, K., & Mattie, H. (2022). Principles of biostatistics. Chapman and Hall/CRC. https://doi.org/10.1201/9780429340512
27. Pathak, D. (2025). Enemies of the hormones: microplastics and endocrine disruptors impacting public health. *Health and Climate Change*, Elsevier, 119–150.
28. Roursgaard, M., Hezareh Rothmann, M., Schulte, J., Karadimou, I., Marinelli, E., & Møller, P. (2022). Genotoxicity of particles from grinded plastic items in Caco-2 and HepG2 cells. *Frontiers in Public Health, 10*, 906430. https://doi.org/10.3389/fpubh.2022.906430
29. Salman, S. A., & AL-Jumaily, R. M. K. (2024). Evaluation of Some Immunological and DNA Damage Parameters among Patients with Rheumatoid Arthritis. *Iraqi Journal of Science.* https://doi.org/0009-0001-0686-4825
30. Santacruz-Juárez, E., Buendia-Corona, R. E., Ramírez, R. E., & Sánchez, C. (2021). Fungal enzymes for the degradation of polyethylene: Molecular docking simulation and biodegradation pathway proposal. *Journal of Hazardous Materials, 411*, 125118. https://doi.org/10.1016/j.jhazmat.2021.125118
31. Sultan, R. S., Al Khayali, B. D. H., Abdulmajeed, G. M., & Al-Rubaii, B. A. L. (2023). Exploring small nucleolar RNA host gene 3 as a therapeutic target in breast cancer through metabolic reprogramming. *Opera Medica et Physiologica*, 10(4), 36-47. https://doi.org/10.24412/2500-2295-2023-4-36-47
32. Sun, A., & Wang, W.-X. (2023). Human exposure to microplastics and its associated health risks. *Environment & Health, 1*(3), 139–149. https://doi.org/10.1021/envhealth.3c00053
33. Sun, H., Chen, N., Yang, X., Xia, Y., & Wu, D. (2021). Effects induced by polyethylene microplastics oral exposure on colon mucin release, inflammation, gut microflora composition and metabolism in mice. *Ecotoxicology and Environmental Safety, 220*, 112340.
34. Tang, K. H. D., Li, R., Li, Z., & Wang, D. (2024). Health risk of human exposure to microplastics: a review. *Environmental Chemistry Letters, 22*(3), 1155–1183.
35. Wei, Y., Zhou, Y., Long, C., Wu, H., Hong, Y., Fu, Y., Wang, J., Wu, Y., Shen, L., & Wei, G. (2021). Polystyrene microplastics disrupt the blood-testis barrier integrity through ROS-Mediated imbalance of mTORC1 and mTORC2. *Environmental Pollution, 289*, 117904. https://doi.org/10.1016/j.envpol.2021.117904
36. Yao, Z., Seong, H. J., & Jang, Y.-S. (2022). Environmental toxicity and decomposition of polyethylene. *Ecotoxicology and Environmental Safety, 242*, 113933. https://doi.org/10.1016/j.ecoenv.2022.113933