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Effects of Oral Polyethylene Microplastic Dose on Wistar Rats' Cognition Assessed by Morris Water Maze

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Keywords

LDPE microplastics; cognitive function; Morris Water Maze; Wistar

Abstract

Orally ingested polyethylene (LDPE) microplastics are suspected to impair cognitive function through disturbances in the central nervous system, particularly in cognitive regions such as the cortex and hippocampus. This study aims to evaluate the effect of varying oral doses of LDPE microplastics on the cognitive function of Wistar Rattus norvegicus using the Morris Water Maze (MWM) test. This experimental study employed a post-test only control group design involving 21 male Wistar rats divided into four groups: a control group and three treatment groups receiving LDPE microplastics at low dose and high dose administered orally for 28 days (particle size ≤20 μm). Cognitive function was assessed using the latency time to locate the hidden platform in the MWM and analyzed statistically. The mean latency tended to increase with increasing dose. Using the control group as the baseline, there was a slight increase in the outcome in the low dose group, and a markedly higher increase observed in the high dose group. Statistical analysis showed a significant difference between the control group and the high dose group ($p = 0.009$). Oral administration of LDPE microplastics at high dose for 28 days was associated with a significant impairment in cognitive function.

INTRODUCTION

Plastic is an essential material in modern life, with global production exceeding 400 million tons annually (Luo et al., 2025). Plastic has become popular because of its versatility, functional diversity, relatively low cost, and light weight (Hesti et al., 2021; Luo et al., 2025). One widely used plastic is polyethylene (PE), which is typically transparent or white, has a melting point of approximately 100-137°C, and is resistant to many chemicals (Rahmawati, 2015). Therefore, polyethylene is widely used in packaging industries, such as shopping bags, bottled beverage containers, food containers, and even automotive components (Yao et al., 2022).

Microplastics are now seen as pollutants that are almost everywhere. They are found across land, freshwater, oceans, and the air, from deserts to farms, and mountaintops to oceans. Land environments are major accumulation areas because most plastics are made, used, and thrown away on land. Plastic waste from agriculture can break down into microplastics and remain in soil for a long time, reported for up to about 15 years (Luo et al., 2025; Pilapitiya & Ratnayake, 2024). Freshwater systems such as rivers, lakes, and urban drainage are also heavily polluted and act as main pathways carrying plastics to the ocean. Once in the water microplastics can be taken up by aquatic organisms (like fish), accumulate in different tissues and organs, and move through the food chain UNEP estimates that nearly 1,000 rivers contribute around 80% of the world's annual river-based plastic emissions to the sea (Cui et al., 2023; Pilapitiya & Ratnayake, 2024). In addition, very small microplastic particles can be carried by wind, especially in cities, and spread over long distances through atmospheric circulation. In the ocean, plastics are found from coastlines to deep

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waters, and they may eventually settle on the deep seafloor, where microplastic pollution has also been detected (Pilapitiya & Ratnayake, 2024).

Microplastics are plastic particles smaller than 5 mm that are formed through the degradation of plastic materials. Based on their origin, microplastics are classified as primary or secondary. Primary microplastics are intentionally manufactured as small particles, while secondary microplastics are formed through environmental degradation of larger plastic items (Luo et al., 2025; Naomi, 2023).

Nevertheless, previous studies indicate that the toxic impacts of microplastic exposure in humans are not yet fully understood (Blackburn & Green, 2022). Other studies have shown that microplastic exposure can trigger oxidative stress and inflammation in the body. In 2019, the WHO reported that humans may ingest approximately 74,000 to 120,000 microplastic particles each year (Luo et al., 2025; Naomi, 2023).

Cognitive function itself refers to a set of mental abilities through which sensory stimuli are received and subsequently processed before being stored, interpreted, and utilized. These capacities enable individuals to aquire, manage, retain, and apply information from the environtment (Hall & Guyton, 2021; Prahasasgita & Lestari, 2023). These function including learning, memory, language, attention, judgement, perception, and executive function such as planning, strategic thinking and problem solving (Pragholapati et al., 2021; Prahasasgita & Lestari, 2023). These process are primarily regulated by the brain, particularly the cerebral cortex (Hall & Guyton, 2021). Importantly, cognitive capacity substantially influences quality of life: cognitive decline commonly observed in older adults may impair self-recognition, the ability to recognize close relatives, and the performance of basic activities of daily living (e.g., eating and personal hygiene), thereby negatively affecting overall quality of life (Pragholapati et al., 2021).

Ingested microplastics enter the gastrointestinal tract and can be taken up through endocytosis by M (microfold) cells in Peyer's patches and by intestinal peristaltic movement. Particles can move from the intestinal lumen into mucosa-associated lymphoid tissue. Microplastics may also be absorbed through gaps in the single-layer epithelium at the villus tips, allowing entry into systemic circulation. Once in the bloodstream, microplastics can distribute widely to various organs in the body (Luo et al., 2025; Naomi, 2023).

The ability of microplastics to penetrate the blood-brain barrier (BBB) is influenced by particle size, chemical properties, binding affinity, and the interacting cell types (Kopatz et al., 2023). This process is more likely when the BBB is compromised, such as during chronic inflammation (Dzierżyński et al., 2024). Biomolecular coronas also play a role: a cholesterol corona can facilitate BBB penetration, whereas a protein corona may inhibit it (Kopatz et al., 2023).

Microplastic accumulation can induce oxidative stress and inflammation (Liu et al., 2022; Luo et al., 2025). An imbalance of reactive oxygen species (ROS) contributes to oxidative stress, which can damage cellular structures, promote lipid peroxidation, and induce programmed cell death or apoptosis (Lee et al., 2022; Naomi, 2023; Sies & Jones, 2020). Antioxidant enzymes may counteract this process (Blackburn & Green, 2022; Liu et al., 2022). A study by Kaur et al. (2024) concluded that antioxidant administration after microplastic exposure can help restore ROS balance, improving learning ability, and reduce anxiety in experimental animals.

Neurotoxicity from microplastics may affect the cortex and hippocampus, which are critical for learning and memory formation. Damage to these regions can impair cognitive function (Hall & Guyton, 2021; Kaur et al., 2024). One method to

assess cognitive function is the Morris Water Maze (MWM), which was developed to evaluate spatial learning and memory in rodents and is widely regarded as a gold standard in behavioral neuroscience (Sharma, 2009). Thus, the MWM can be used to assess hippocampal and cortical function; cognitive impairment is indicated by increased latency to locate the hidden platform (Nunez, 2008; Sharma, 2009).

Therefore, this study aimed to determine whether oral administration of polyethylene microplastics at various doses affects cognitive function in male Wistar rats (Rattus norvegicus) as assessed by the MWM. If confirmed, this study may help identify a minimum dose threshold that could be considered safe with respect to cognitive function.

RESEARCH METHODS

Data Collection Procedure

This was a quantitative experimental study using a post-test-only control group design. The study was conducted at the Animal Laboratory and Research Laboratory, Faculty of Pharmacy, Widya Mandala Catholic University Surabaya. Animal termination was performed at the Pharmacology Laboratory, Faculty of Medicine, Widya Mandala Catholic University Surabaya. The study was carried out from 21 July to 26 August 2025. The subjects were 21 male Wistar rats (Rattus norvegicus), aged 6-8 weeks, with body weights of 150-200 g. All rats were randomly allocated into four groups and labeled X0 (control), X1 (low dose: 1.25 mg/day), X2 (high dose: 2.5 mg/day), with 7 rats per group.

After random allocation, all rats underwent an 8-day acclimatization period and were housed individually (one rat per cage). Rats received Fur594 feed (30 g per rat) and mineral water ad libitum, replaced daily. After acclimatization, treatments were administered for 28 days. The control group (X0) received distilled water (aquabides) by oral gavage, whereas the treatment groups (X1, and X2) received a microplastic suspension in distilled water according to the assigned dose. Gavage was performed once daily in all groups with a total volume of 1 cc.

Microplastic Characteristics

The microplastics were obtained from an LDPE pipe that was mechanically ground using a BOSCH CWS 700 grinder and then sieved using an 800-mesh sieve (18 µm pore size) to obtain particles ≤20 µm. The microplastic preparation was observed under a Nikon Eclipse Ci microscope (40×10 magnification), and the LDPE polymer type was confirmed by FTIR analysis. The microplastic suspension was stored in a tightly closed glass bottle at room temperature and protected from direct light. Before administration, the suspension was shaken to ensure dispersion.

Cognitive Function Assessment

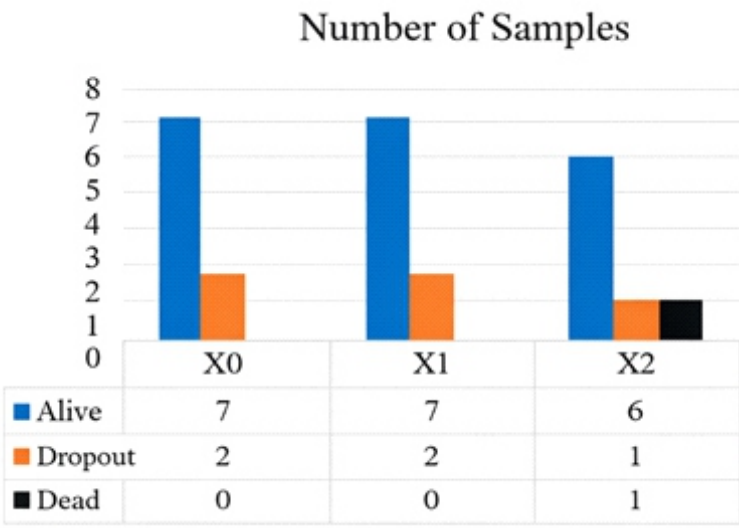
Cognitive function was assessed using the MWM with a circular pool 80 cm in diameter, water depth of approximately 20 cm, and a wooden platform measuring 10×10 cm. After acclimatization, pre-training was conducted using clear water and a visible platform (1 inch above the water surface). Testing was performed after the 28-day treatment period using water made opaque with non-toxic paint and a platform placed 0.5-1.0 cm below the surface. Each rat completed 12 trials over 2 days (6 trials/day), with 3 trials from each quadrant (north, south, east, and west).

Data Analysis

Data were analyzed using SPSS version 27. Normality was assessed using the Shapiro-Wilk test and homogeneity using Levene's test. Between-group comparisons were performed using one-way ANOVA for homogeneous data; otherwise, the Kruskal-Wallis test was used. Post-hoc analyses used the LSD-test (for homogeneous data) or the Mann-Whitney test (for non-homogeneous data).

RESULTS AND DISCUSSION

Figure 1 shows the survival status of rats during the experiment, categorized as alive, dropout, and dead across the control group, the low-dose treatment group, and



Source: Processed Primary Data, (2025)

Figure 1
Number of Rats at the End of the Treatment Period
Note: X0= control rat, X1: rats with low dose, X2: rats with high dose

the high-dose treatment group. This figure provides an overview of the distribution of rats in each group to illustrate the survival condition and any changes observed during the study period.

Based on Figure 1, the number of rats classified as alive, dropout, and dead among control rats, rats with a low dose, and rats with a high dose. In the control rat group, there were 7 rats alive, 2 dropout, and no deaths recorded. A similar pattern was observed in rats with a low dose, where 7 rats remained alive, 2 were categorized as dropout, and no mortality occurred. In contrast, in rats with a high dose, the number of alive rats slightly decreased to 6, the dropout decreased to 1, and 1 rat was recorded as dead. Overall, the results indicate that survival remained relatively high across all groups, although a slight mortality appeared in the high-dose treatment group.

After the 28-day treatment period, MWM data were collected from all remaining animals. Testing was conducted over 2 days (6 trials/day). The results are presented in Table 1, with the summary is shown in Table 2.

Table 1
Mean MWM Latency Result for Each Rat

Rats Group		Mean MWM latency (s)
Control	1	6.08
	2	6.98
	3	9.45
	4	5.42
	5	4.91
Low dose	1	15.86
	2	6.93
	3	9.47
	4	6.94
	5	5.22
High dose	1	9.58
	2	9.92
	3	10.22
	4	10.37
	5	26.68

Source: Processed Primary Data, 2025

Table 2
Summary Result of MWM Latency

Group	Mean latency (s) ± SD	Difference vs control (s)	Percentage increas vs X0
Control	6.57 ± 1.78	0	0
Low dose	8.88 ± 4.18	2.31	35.16%
High dose	13.39 ± 7.53	6.82	103.81%

Source: Processed Primary Data, 2025

Based on Table 1, the mean MWM latency of rats varied across the three groups. In the control group, the latency values ranged from 4.91 to 9.45 seconds, indicating relatively faster platform-finding performance. In the low-dose group, the latency values ranged from 5.22 to 15.86 seconds, showing slightly higher variability and generally longer times compared to the control group. Meanwhile, the high-dose group showed the highest latency values, ranging from 9.58 to 26.68 seconds, with one rat demonstrating a markedly prolonged latency.

Table 2 summarizes the MWM latency across the experimental groups and reveals patterns that are not immediately apparent from the raw data. Although the control group showed a relatively low mean latency (6.57 ± 1.78 s), indicating consistent spatial learning performance, the low-dose group demonstrated not only a higher mean latency (8.88 ± 4.18 s) but also a noticeably larger standard deviation. This suggests that the effect of the low dose was not uniform among rats, with some individuals showing performance similar to the control while others experienced delays in locating the platform. The high-dose group exhibited both the highest mean latency (13.39 ± 7.53 s) and the greatest variability, indicating that exposure at this level may substantially disrupt spatial learning or memory in certain rats.

Statistical Analysis Result

The Shapiro-Wilk normality test indicated that the overall data were not normally distributed; therefore, subsequent analyses used non-parametric tests. Subsequently, comparative analyses were conducted between the control group and each experimental group.

The Kruskal-Wallis test among control, low dose, and high dose group showed a significant difference ($p<0.05$). Post-hoc Mann-Whitney comparisons with the control group showed a significant difference for high dose group ($p<0.05$), indicating a significant decline in cognitive function at the high dose.

In this study, 28 days of oral exposure to polyethylene (LDPE) microplastics showed a dose-related tendency toward increased MWM latency, with increases of 35.16%, and 103.81 relative to controls. Increased latency to locate the hidden platform is commonly used as an indicator of reduced cognitive function. The Shapiro-Wilk test suggested a non-normal distribution; thus, non-parametric analysis was applied. The overall Kruskal-Wallis comparison across all groups show a significant difference.

Table 3
Normality Test

Grou p	Normality Test	P
Control	*Saphiro -Wilk*	0.417
Low dose		0.175
High dose		0.001

Source: Processed Primary Data, 2025

Tabel 2
Summary of Willingness to Pay Estimation for Existence Value

Variabl e	Group	Comparative Test	P	Conclusion
Cognitive Function MWM	Control , Low dose , and H igh dose group	*Kruskal -Wallis*	0.024	A statistically significant
	Con trol - Low dose	*Mann -Whitney*	0.374	No statistically significant
	Control - High dose		0.009	A statistically significant

Source: Processed Primary Data, 2025

Mann-Whitney analysis revealed a more specific pattern: a significant difference between the control group and the high-dose group. In contrast, the low-dose group were not significantly different from controls. This suggests that, in this study, the most consistent statistically significant decline in cognitive performance occurred at the high dose.

In the low-dose group, micro-plastic exposure may have begun to increase ROS production and induce mild oxidative stress in the cortex and hippocampus; however, this may still have been compensated for by endogenous antioxidant systems (e.g., superoxide dismutase, catalase, and glutathione peroxidase). Therefore, if antioxidant capacity is still able to counterbalance the increase in ROS, neuronal damage has not yet become extensive enough to cause a significant decline in cognitive function (Albano et al., 2022; Hong et al., 2024; Sies & Jones, 2020). In the high-dose group, the amount of microplastics reaching the cortex and hippocampus may have been sufficient for ROS production to exceed antioxidant capacity, leading to oxidative stress that can damage proteins and DNA and trigger neuronal apoptosis, particularly in learning and memory regions such as the hippocampus, thereby disrupting learning ability and memory consolidation (Albano et al., 2022; Kaur et al., 2024; Sies & Jones, 2020).

Biologically, the plausibility of microplastic-associated cognitive decline is supported by the oral exposure pathway Ingested microplastics can be absorbed via M-cell endocytosis in Peyer's patches and by persorption through epithelial gaps at villus tips, enter systemic circulation, and distribute to organs including the brain (Luo et al., 2025; Palyama et al., 2023). Once in the brain, microplastics may accumulate in the cortex and hippocampus and potentially injure neurons (Kaur et al., 2024; Liu et al., 2022). Microplastics may cross the BBB through mechanisms influenced by particle size, chemical properties, binding affinity, and interacting cell types (Kopatz et al., 2023). BBB penetration may be more likely when the BBB is damaged, such as due to chronic inflammation (Dzierżyński et al., 2024). Biomolecular coronas may also influence BBB penetration: a cholesterol corona can facilitate penetration, whereas a protein corona may inhibit it (Kopatz et al., 2023).

These findings are in line with a previous study by Kaur et al. (2024), which used polystyrene microplastics. Kaur et al. (2024) reported that oral gavage exposure to 2-µm polystyrene microplastics increased MWM latency, with increases observed after 15 days and becoming more pronounced with longer exposure. The study also concluded that microplastics can cross the BBB, accumulate in the brain, and contribute to increased anxiety and decreased learning ability.

Lee et al. (2022) demonstrated that oral exposure to polystyrene microplastics can impair learning, resulting in higher latency than controls.Differences in polymer type, dosing regimen, and

individual response variability may help explain why the overall analysis in the present study was not significant despite a clear trend toward increased latency.

Finally, interpretation of these results should consider the study limitations. Histopathological analyses of brain tissue and blood were not performed to confirm microplastic accumulation and to clarify mechanisms in detail. Assessment was conducted at a single time point, so acute and chronic dynamics were not captured, and potential confounders that could influence microplastic effects may not have been fully controlled. Future studies incorporating histopathology, oxidative stress and inflammatory biomarkers, and cognitive testing at multiple time points would help clarify the dose-response relationship and underlying mechanisms of microplastic neurotoxicity.

CONCLUSION

In this post-test only experimental study, male Wistar rats were acclimatized for 8 days, and then exposed for 28 days to daily oral gavage of LDPE microplastic (≤20µm) at low and high dose. While control received distilled water. Cognitive performance was assessed after treatment using the Morris Water Maze following pre-training with a visible platform. Overall, the MWM escape latency indicates that higher doses of microplastics are associated with longer latency times. This pattern suggests a tendency for reduced spatial learning and memory performance in groups exposed to higher doses of microplastics compared to the control group. Non-parametric testing identified a significant difference specifically between the control group and the high dose group, whereas comparisons with the low-dose groups were not significant. Biologically, these behavioral findings align with the study's proposed mechanism: orally ingested microplastics may enter systemic circulation and potentially reach the brain, where they can contribute to oxidative stress and neuroinflammatory injury in cognition-related regions (coretax and hippocampus). The pattern in this dataset suggests that low-dose exposure may still be partially compensated by endogenous antioxidant defenses, the high dose may exceed compensatory capacity leading to more consistent impairment, and higher-dose exposure may produce heterogeneous responses across individuals. Given the absence of histopathology or biomarker confirmation and the single post-treatment time point, further work should include larger samples, verification of microplastic accumulation and oxidative or inflammatory markers, and repeated cognitive assessments across time to better define the dose response threshold for LDPE microplastic neurotoxicity.

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